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THE UNIVERSITY OF ALBERTA

THE CARDIOVASCULAR RESPONSE OF POST CORONARY ARTERY BYPASS SURGERY PATIENTS TO HYDRAULIC CIRCUIT TRAINING

BY

©

ROBERT GERALD HAENNEL

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

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FALL, 1987

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Reseach for acceptance, a thesis entitled THE CARDIOVASCULAR RESPONSE OF POST CORONARY ARTERY BYPASS SURGERY PATIENTS TO HYDRAULIC CIRCUIT TRAINING submitted by Robert Gerald Haennel in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Physical Education and Sport Studies.

Quinney

Supervisor

A. Belcastro

Kappagoda

David Magee

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Date June 2, 1987

DEDICATION

TO MY FAMILY

ABSTRACT

This series of studies was undertaken to determine the feasibility of hydraulic circuit training (HCT) as an alternate training mode in cardiac rehabilitation. Prior to investigating the effects of HCT in patients recovering from coronary artery bypass surgery (CABS) the acute cardiovascular (CV) response to isokinetic exercises (ISOK EX), was assessed in 5 healthy male subjects who performed unilateral knee and elbow extension/flexion exercises (ELBOW), at velocities of, 0.52, 1.57, and 2.62 $rad \cdot s^{-1}$. In response to knee extension/flexion exercises (KNEE) HR, CO and mean arterial pressure (MAP) increased ($p<0.01$). The CO and MAP responses were not influenced by the exercise velocity. The changes in HR and MAP observed during the ELBOW were qualitatively similar to those seen during the KNEE but the actual changes were smaller. Results suggest that, during ISOK EX the increase in HR and MAP is related to the active muscle mass and independent of the exercise velocity.

The effects of HCT on CV function was then assessed in 32 healthy middle-aged males. Twenty-four men were randomly assigned to a control (CG) or one of two HCT groups. Both HCT groups participated in an 8 week training program. One HCT group (HCT_{max}) completed the maximal number of repetitions possible (RM) during each 20s work interval. The other HCT group (HCT_{sub}) completed 70-85% of RM for each work interval. Results were compared with 8 men who completed a cycle training program. Following training VO_2 max was increased in all training groups ($p<0.05$). The increase was associated with increases in maximal SV and CO ($p<0.05$). The CV adaptations achieved in the HCT_{sub} group were associated with a lower training HR, and rate pressure product ($p<0.05$). These finding suggest that HCT_{sub} is effective in improving maximal aerobic power at a reduced CV stress.

The effect of HCT was then assessed in 18 CABS patients. Twelve patients were randomly assigned to either 8 weeks of cycling or HCT. Their results were compared with 6 patients who served as a non-exercising control group. Post-training, the cycle and HCT groups demonstrated a similar increase in VO_2 max ($p<0.05$). This increase was associated with increases in maximal SV, and CO, with a relative bradycardia at submaximal workloads. Results suggest that HCT may be a viable training modality for CABS patients who can only tolerate a limited amount of cardiovascular stress.

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CHAPTER 1

INTRODUCTION

From the viewpoint of the World Health Organization, the primary objective of cardiac rehabilitation is to prepare the individual for a return to an optimal physical, vocational, and recreational status (11). Over the past 30 years exercise training has gained widespread acceptance as an integral component of this rehabilitative process. Conventional training programs are aerobic in nature and incorporate activites such as walking, jogging and cycling. While there is little doubt that aerobic training will enhance systemic oxygen uptake (2,17), such training precipitates large increases in heart rate and blood pressure, thereby placing heavy demands on the central circulation. Patients with more severe disease are less able to tolerate vigorous aerobic exercise because of symptoms. Moreover, these patients are at risk of ischemia-induced arrhythmias while engaging in aerobic type activities of intensity sufficient to produce central and peripheral training effects seen in healthy individuals (3).

For an exercise training program to be both safe and effective for these cardiac patients the regimen should incorporate interval training which focuses on smaller muscle masses and relatively high intensities. Such a program may achieve the peripheral adaptations associated with training while, at the same time, avoiding the heavy sustained demands on the central circulation associated with aerobic exercises. A regimen which may meet these requirements is a circuit training program utilizing hydraulic resistance equipment. There are several practical advantages to hydraulic circuit training. First, the equipment is capable of low power outputs with smaller muscle groups, which should limit the demand for an increase in myocardial oxygen uptake during the actual exercise. Second, hydraulic resistance devices work the agonist and antagonist muscle groups concurrently, and accommodate to the force produced over the range of motion, thus optimizing both exercise time and intensity. Another advantage of hydraulic resistance devices, over more conventional free weights and pully systems, relates to the exercise skill requirement. Hydraulic resistance devices are passive, thus minimizing the risk of muscular and joint strain which can result from efforts to control an applied force (such as lifting or lowering a weight). Finally hydraulic circuit training is performed in an interval fashion, so as to attenuate the need for a sustained stress on the cardiovascular system.

The nature of occupational stresses, and the absence of significant cardiovascular and metabolic crossover effect with aerobic training (9) suggests a practical benefit to hydraulic circuit training in cardiac rehabilitation. Studies of a large number of subjects in a variety of occupations indicate that the physical stresses of occupational energy expenditure are usually low, involving submaximal muscular effort of brief duration (i.e. intermittent and not continuous). These early studies (7,8) revealed that the major muscle groups of the upper extremeties, rather than the lower extremeties, are called upon extensively, and that few occupations required sustained walking or jogging (9). Thus, factors other than aerobic energy expenditure constitute the major occupational stresses.

Based upon a need to maintain or improve muscular strength and endurance, an examination of hydraulic circuit training as component of cardiac rehabilitation is warranted. Traditionally, muscular strength and endurance exercises were not included in cardiac rehabilitation, because it was felt that they produce ischemic changes and increased myocardial irritability (1). However, subsequent investigations have been unable to corroborate these earlier results (4,16,19,20). Moreover, recent studies have demonstrated the relative safety of weight carrying and circuit weight training in selected cardiac patients (16,18,21). The significant increase in blood pressure usually associated with strength training exercises appears to be a function of the muscle mass and neural innervation, rather than a question of the nature of the exercise (14,15). Further, a concomitant valsalva maneuver may actually account for a significant portion of the increased blood pressure usually associated with strength training exercises (12).

Hydraulic circuit training has some merit for coronary patients who want to return to occupations requiring moderate manual labor or frequent lifting. An individual whose vocational and avocational activities involves mostly upper body muscular exertion should train those muscle groups, with the expectation that the cardiovascular stress associated with these activities will be reduced following training (6,9).

In addition to the clinical interests of hydraulic circuit training, it is also of physiological interest to evaluate to what extent cardiovascular parameters can be altered by training which does not produce sustained central circulatory loading. Preliminary studies in healthy subjects should be carried out to evaluate both the practical and physiological aspects of hydraulic circuit training.

Thus the purpose of the present study was to:

1. Investigate the cardiac output and blood pressure changes associated with maximal isokinetic exercise.
2. To determine whether a circuit training program using hydraulic resistance equipment will enhance the maximal aerobic power of previously untrained middle-aged men.
3. To document the heart rate and cardiac output response of a group of post coronary artery-bypass surgery patients to maximal hydraulic resistance exercise.
4. To examine the efficacy of a hydraulic circuit training program on the maximal aerobic power of post coronary artery-bypass surgery patients.

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CHAPTER 2

GENERAL METHODS

Measurement of Stroke Volume and Cardiac Output

In this series of investigations, stroke volume and cardiac output were measured using the Minnesota Impedance Cardiograph Model 304A (Surcom Inc., Minneapolis). Impedance Cardiography is a non-invasive, atraumatic method of measuring heart rate, stroke volume and consequently cardiac output. The technique involves the placement of four electrode bands around the neck and thorax. A weak frequency alternating current is passed though the outer two electrodes. The constant alternating current is so weak that it is imperceptible to the subject and the frequency is so high it is incapable of stimulating the heart (7).

The use of this technique has been validated in this laboratory for endurance trained young male subjects and cardiac patients, both at rest and during exercise (4,15). The systemic error of the technique was assessed over the range 3.5 to 18 $l \cdot min^{-1}$ by comparing with simultaneous measurements of cardiac output made using the direct Fick method. No systemic error was demonstrated in 40 estimations made in 20 subjects (4). The random error, assessed at rest and during steady state exercise was less than 5%. The reproducibility of the maximal exercise response was assessed in subjects who underwent 2 maximal exercise tests one week apart. Highly significant correlations were obtained for both stroke volume

($r = 0.84$, $p<0.001$) and cardiac output ($r = 0.98$, $p<0.001$) between the 2 tests (15).

Procedure. On the day of the investigation the subjects reported to the laboratory in a fasting state. The four bands of self-adhesive disposable mylar-backed aluminum electrode tape were placed around the neck and chest 30 minutes before commencement of the study. Two bands were placed around the neck, 3 to 5 cm apart, the third around the trunk at the level of the xiphisternum and the fourth at a level just above the umbilicus. When connected the two outer electrodes transmitted a constant sinusoidal alternating current (4 ma RMS and 100 KHz) through the thorax and the changes in transthoracic electrical impedance were detected by the inner two electrodes (Fig. 2.1A). Mean total transthoracic impedance between the inner electrodes (Z_o) was computed by, and displayed on the Impedance Cardiograph. Simultaneous recordings of the rate of change of impedance through each phase of the cardiac cycle (dZ/dt), the electrocardiogram and phonocardiogram were made on a 4 channel ink recorder at a paper speed of 50 $mm \cdot s^{-1}$ (Model 2400S, Gould Inc., Cleveland). The heart rate, dZ/dt_{min}, and left ventricular ejection time, were obtained from the recordings as shown in Fig 2.1B. Calculations of stroke volume was made using the following equation (8):

$$\text{Stroke Volume} = \frac{P \times L^2 \times dZ/dt_{min} \times T}{Z_o^{\ 2}}$$

where

P = electrical resistivity of blood at body temperature

$(P = 53.2e^{0.022H})$ (3),

H = hematocrit (%),

L = average distance (cm) between the inner pair of electrodes measured at the anterior and posterior midline,

Z_o = mean transthoracic impedance (ohms) between the inner two electrodes,

dZ/dt_{min} = minimum value for the rate of change of impedance (ohms) occuring during the cardiac cycle,

T = left ventricular ejection time (s).

Recordings were made at rest and during exercise. It was found that movement caused by respiration and exercise introduced artifacts into the recordings. These artifiacts were avoided by requesting the subjects to stop all movement, remain motionless and hold their breath at normal end-expiration for approximately 5s, while 5 to 10 cardiac cycles were recorded. An average of 5 cardiac cycles were used for the calculation of stroke volume. The hematocrit was measured from blood samples drawn from an antecubital vein immediately following each recording.

Assesssment of Maximal Aerobic Power

Maximal aerobic power was assessed by measuring the maximum oxygen uptake (VO_2 max) achieved during a Graded Exercise Test (GXT) on a bicycle ergometer (Model 740, Siemans Electric Ltd., Missassuaga) in the upright position. All subjects came to the laboratory in a fasting state and were familiar with the apparatus prior to the assessment. Unless otherwise indicated the initial workload was 30 watts with 20 watts increments every three minutes.

The VO_2 was measured at each workload using a continuous flow technique (1). The highest VO_2 attained during the GXT was recorded as VO_2 max. Blood pressure was measured using a mercury sphygmomanometer at rest and at the end of the second minute of each workload during the GXT. A 12-lead electrocardiogram was monitored continuously thoughout the GXT. The GXT continued until one or more of the following end points was achieved: a leveling off of, or decrease in VO_2 with increasing workloads; attainment of 90% age predicted maximal heart rate; chest pain; electrocardiographic ST-segment changes compatible with ischemia (i.e. >2mm depression); ventricular arrythmias during exercise (i.e. PVC>10/min, multifocal PVC's, couplets or ventricular tachycardia); an abnormal drop in blood pressure, or a failure of the blood pressure to increases over three consecutive workloads; volitional exhaustion (Borg scale reading >18); dizziness; or shortness of breath (14).

Measurement of Muscular Strength and Endurance

A Cybex II Isokinetic dynamometer (Lumex, Inc., Ronokonkoma, New York) equipped with a two channel recorder was used to measure muscular strength and endurance. The isokinetic dynamometer allows voluntary contractions to be made at various pre-determined constant velocities regardless of the magnitude of the forces generated by the participating muscles (11). Acceleration occurs until the pre-determined velocity is achieved by the exercising limb. Once this velocity is reached, the isokinetic apparatus resists further acceleration and the excess force applied by the subject to the lever arm of the dynamometer is recorded as torque (5). Moffroid et al

(10), and Thorstensson et al (16) have reported reliability coefficients for torque outputs as high as 0.995 and validity coefficients between predicted and obtained power of 0.990. To ensure constant velocity of the lever arm thoughout the entire range of motion, a reliability correlation of 0.985 was reported for 32 points on generated torque curves (10).

Procedure. Measurements were made during a series of maximal extension/flexion exercises. The subjects were familarized with the system and the required limb actions prior to the assessments. Limb alignment and stabilization procedures were standardized for each test (2). In order to minimize artifactual torques, or the torque "overshoot" the first extension and flexion torques from each series were omitted from the results, and an optimal damping setting of 2 on the Cybex II chart recorder was used (13). Muscular strength was defined as the peak torque (N·m) recorded during a given exercise. The assessment of muscular strength was completed at two velocities, 1.05 and 3.14 $rad \cdot s^{-1}$. Muscular endurance was defined as the total work accummulated (expressed in kilojoules, kJ) over three 20s bout of maximal exercise at 3.14 $rad \cdot s^{-1}$, and was estimated by calculating the average torque (the area under the torque curve, in $N \cdot m \cdot s^{-1}$ divided by the contraction time, in s) and then multiplying it by the angular displacement (rads) (12). In all cases the 1.05 $rad \cdot s^{-1}$ test was conducted first, with a minimum 5 minute recovery interval before the 3.14 $rad \cdot s^{-1}$ test.

Hydraulic Resistance Exercise

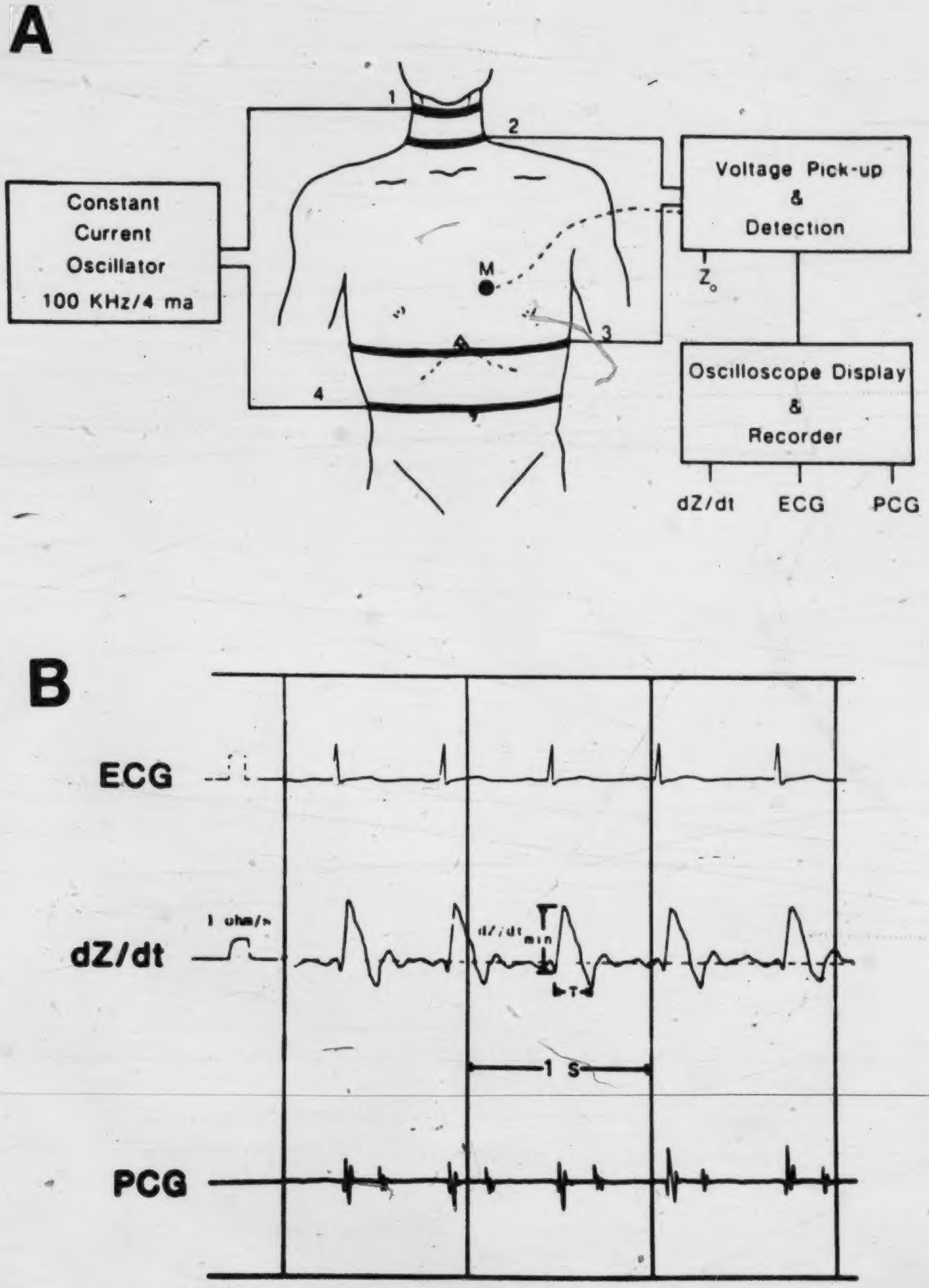
For the circuit training programs the subjects exercised on devices that provided resistance from variable hydraulic cylinders (Hydra-Fitness Canada Ltd., Sherwood Park Alberta). These hydraulic cylinders allow both variable speed and resitance over the range of motion (9). An important design feature of this equipment is that it permits concentric-only exercise for the agonist and antagonist muscle groups during completion of a given movement. The resistance to movement can be regulated by selecting one of six valve settings on the machine. These settings correspond to six orifice sizes though which the hydraulic fluid passes. The diameter of the orifaces vary from 0.076 mm (setting 1) to 0.031 mm (setting 6) (6).

For the present investigations, hydraulic resistance equipment was incorporated into a circuit training program wherein the subjects performed three sets of each exercise, with 20s work intervals and a 1:2 work:relief interval. Whenever possible the exercises were ordered so as to alternate upper and lower body movements.

Figure 2.1A and B

A. Electrode Placement used to record Thoracic Impedance Cardiogram: Attachment of strip electrodes on a subject; electrodes 1 and 4 are connected to the constant current oscillator while electrodes 2 and 3 are used to detect changes in current. (Z_o: average total transthorasic impedance, dZ/dt: rate of change of impedance, ECG: electrocardiogram, PCG: phonocardiogram, M: microphone placed on the precordium).

B. Typical impedance cardiogram recording with simultaneous ECG and PCG. Measurement of dZ/dt_{min} and ejection time (T) is as shown. Broken horizontal line shown with the dZ/dt record is the calibration baseline.


A
1
2
3
4
M
Constant Current Oscillator 100 KHz/4 ma
Voltage Pick-up & Detection
Z₀
Oscilloscope Display & Recorder
dZ/dt
ECG
PCG
B
ECG
1 ohm/s
dZ/dt
dZ/dt min
T
1 s
PCG

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CHAPTER 3

CHANGES IN CARDIAC OUTPUT AND BLOOD PRESSURE DURING ISOKINETIC EXERCISE*

Introduction

Studies on the cardiovascular response to exercise have focused on an understanding of the effects of isometric and dynamic activity. An isometric activity denotes muscular contractions in which force is produced with little or no change in the angle of the joint. Dynamic activity involves isotonic muscular contractions against mechanical systems which provide a constant load. During dynamic activity the body segments move against this load through a range of motion, as when lifting a weight or overcoming a braked resistance. Although the external load is constant during an isotonic contraction, the muscle is presented with an ever-changing resistance over the range of motion because of the modifying effect of the lever system (8). A dynamic activity wherein the loading of the muscle is maximal throughout the range of motion is referred to as isokinetic exercise.

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The unique feature of isokinetic exercise is the control of the velocity of muscular performance (16,17). An isokinetic dynamometer allows voluntary contractions to be made at various pre-determined constant velocities regardless of the magnitude of the forces generated by the participating muscles. Acceleration occurs until the pre-determined velocity is achieved by the exercising limb. Once this velocity is reached, the isokinetic apparatus resists further acceleration and the excess force applied by the subject to the lever arm of the dynamometer is recorded as torque (8). Therefore an isokinetic exercise differs from an isometric and isotonic contraction in that the muscle contracts against an accommodating resistance that allows only a constant rate of movement while receiving the full muscular force of the participating muscles throughout the range of motion (18).

Although several investigators have reported on the cardiovascular responses to isometric exercise involving small and large muscle masses (5,10,14,20), as well as dynamic exercises such as cycling (4,9) and weight lifting (7,12), the corresponding responses to isokinetic exercise have not been fully documented. The purpose of this study was to investigate the cardiac output and blood pressure changes associated with maximal isokinetic exercise, and to compare these changes with those associated with maximal isometric exercise. As the mode of contraction was expected to influence the cardiovascular adjustments data from normal subjects who performed maximal dynamic exercise was included for comparison.

Methods

1) Subjects

The subjects were healthy young adult male volunteers. They were fully informed of the purpose of the experiment and written consent was obtained prior to the study. The cardiovascular response to maximal dynamic exercise was conducted on six subjects. Five subjects participated in the assessment of the acute cardiovascular response to isokinetic exercise. The study protocol was approved by the institutional ethics review committee.

2) Measurement of Work

The isokinetic apparatus used in the present study was a Cybex II Dynamometer, (Lumex, Inc., Ronokonkoma, New York) which was equipped with a two channel recorder. The Cybex II dynamometer allows torque to be applied and measured in opposing directions. In the present study the Cybex dynamometer was used to measure the torque generated during a series of unilateral knee extension/flexion exercises and a series of unilateral elbow extension/flexion exercises. During each exercise bout the subjects were instructed to apply maximum force over the entire range of motion. The normal response consists of two or three torque curves of similar amplitude followed by a gradual degradation of peak torque height (Figure 3.1).

Work (kJ) is the product of force exerted though a specific range of motion (18). The external work completed during each contraction was estimated by calculating the average torque (the area under the torque curve, in $N \cdot m \cdot s^{-1}$, divided by the contraction time, in s), and multiplying by the angular displacement (rads) (18). The values reported represent the external work recorded over each one minute exercise period.

3) Measurement of Cardiac Output

For all the subjects participating in the study stroke volume (SV), heart rate (HR), and cardiac output (CO) were measured by impedance cardiography (Minnesota Impedance Cardiograph, model 304A, Surcom Inc., Minneapolis, Minnesota). The use of this technique has been validated in this laboratory for endurance trained young male subjects and cardiac patients, both at rest and during exercise in a manner described previously (23).

Readings were obtained immediately, (within 3s) upon completion of a given exercise bout. During the measurements, subjects were required to hold their breath at normal end-expiration to avoid artifact due to respiratory movement. The average of five cardiac cycles were used in the calculation of SV.

4) Measurement of Blood Pressure

Once preparation for impedance cardiography was completed those subjects participating in the isokinetic and isometric exercises were positioned on a Cybex knee testing table. The area of the left arm over the radial pulse was prepared with povidone-iodine (Betadine), 2% lidocaine was infiltrated over the radial artery. A cannula was inserted percutaneously into the radial artery (16 gauge 2.25 inch Jelco; Critikon Ltd., Markham, Ontatio). The cannula was connected by a 36 cm extension tube to a pressure transducer (Statham model P23D6; Siemens Electric Ltd., Missassuaga, Ontario) and kept patent with heparinized saline. The pressure transducer was positioned at the level of the fourth intercostal space for each subject. The baseline for the transducer was reset and rebalanced for each subject. Arterial pressures were monitored continuously and recorded using an ink writing system (Mingograph recorder, model 804; Siemens Electic Ltd., Missassauga, Ontario).

Mean arterial pressure (MAP) was calculated as diastolic pressure plus one-third pulse pressure. The MAP reported here are the peak values recorded during each bout of exercise. Systemic vascular resistance (SVR) was calculated as (MAP/CO) x 80 (the factor for converting to $dyn \cdot s \cdot cm^{-5}$), using MAP as the systemic circulation gradient, and assuming the right atrial pressure as zero and not changing from rest to exercise (3). The rate pressure product (RPP) was calculated from HR and systolic blood pressure data ($RPP = HR \times SBP \times 10^{-3}$). Throughout each exercise bout subjects were coached in maintaining a normal rhythmical breathing pattern, to minimize the respiratory influences on right atrial pressure. During all exercise bouts the canulated arm was supported and immobilized and did not contribute to the mechanical work done.

5) Exercise Protocol

The protocol involved three exercise modes; a) isokinetic; b) isometric; and c) dynamic. All exercise bouts were performed at maximal intensity.

a) Isokinetic Exercises. Over the two weeks immediately preceeding the investigation the subjects participated in a series of six preliminary sessions to familiarize themselves with the equipment and the exercise protocol. Two movement patterns were tested, right knee extension/flexion, and right elbow extension/flexion. The exercises were performed at velocities of; 0.52; 1.57; and 2.62 $rad \cdot s^{-1}$. For each movement pattern the exercise velocities were assigned randomly. For both movement patterns, limb alignment and stabilization procedures were standardized. The input shaft of the dynamometer was placed parellel to the limb with the axis of rotation coinciding as closely as possible with the axis of rotation of the joint. For the knee extension/flexion exercises, the subjects were held in position by restraining straps around the waist, thigh, and right knee proximal to the ankle. The latter strap attached the subjects to the lever arm of the isokinetic apparatus. The position of the back rest of the isokinetic table was adjusted so that the angle of the hip was between 1.92 and 2.10 rads (Figure 3.2). For the elbow extension/flexion exercises the subjects were placed supine on the Upper Body Extremety Testing table and secured to the table by restraining straps around the chest and waist. The subjects grasped the handle attatched to the lever arm of the dynamometer when performing the exercise. During each exercise bout the subjects executed the maximal number of movements possible within a one minute period. The recordings of the peak torque curves were examined to ensure that the subjects had exerted a maximum effort. Upon completion of each exercise bout, external work was computed. Each bout of isokinetic exercise was followed by a 5 minute recovery period.

b) Isometric Exercises. The Cybex II dynamometer was also used for the isometric exercises. The subjects performed maximal knee extension and maximal elbow extension exercises. For both exercises the angle of the joint was set at 1.05 rads of flexion (full extension considered 0 rads), the subjects then performed a one minute maximal isometric contraction. To do the test the angle of the limb was fixed and the Cybex velocity selector was set at 0 $rad \cdot s^{-1}$.

c) Dynamic Exercise. The subjects were exercised in an upright posture on a bicycle ergometer (Model 38B Siemans Electric Ltd., Missassuaga, Ontario). The initial load was set at 30 watts (W). At 3-minute intervals the workload was increased successively to 50, and 80 W, and in steps of 50 W thereafter. The increments in work were continued until maximal oxygen uptake (VO_2 max) was achieved as indicated by a plateau in VO_2, which was recorded at the end of each stage of exercise. The SV and CO were measured at the end of each three minute exercise stage up to the maximal load. The BP was measured using a mercury sphygmomanometer during the second minute of each stage of exercise.

6) Statistical Analysis

The data presented are expressed as means ± standard error of means (SEM). Statistical comparison of the changes in the various cardiovascular parameters across the exercise velocities and the two active muscle groups was accomplished by a two-way analysis of variance with repeated measures (2). For all tests a difference was considered statistically significant if $p<0.05$. For all variables in which a significant F value was obtained, a test of Least Significant Difference was performed to assess the significance of the specific differences among the mean values (2).

Results

The study was completed on 11 healthy males (20-33 years). The isometric and isokinetic exercise bouts were performed by 5 male subjects. Mean values for their physical characteristics were: age, 24.6 ± 2.1 yrs; height 178.9 ± 4.1 cm; weight 83.5 ± 3.2 kg. The VO_2 max of this group assessed on a bicycle ergometer was 4.3 ± 0.5 $l \cdot min^{-1}$. Six volunteers (mean age 26.2 ± 4.4 yrs; weight 86.4 ± 4.7 kg; height 180.1 ± 8.7 cm) performed the maximal dynamic exercise bout.

Dynamic Exercise. The average maximal HR attained during the dynamic exercise bout was 185.0 ± 5.6 $beats \cdot min^{-1}$. The VO_2 max was 4.4 ± 0.2 $l \cdot min^{-1}$. At maximal exercise SV averaged 140.3 ± 4.0 $ml \cdot beat^{-1}$. The maximal CO achieved was 26.1 ± 1.6 $l \cdot min^{-1}$. At maximal exercise, MAP was 121 ± 5 mmHg, compared to the pre exercise value of 88 ± 2 mmHg ($p<0.05$). The SVR decreased from a pre exercise level of 1168 ± 56 to 392 ± 32 $dyn \cdot s \cdot cm^{-5}$, at maximal exercise ($p<0.05$). At maximal exercise the RPP was 31.5 ± 8.3 (HR x SBP x 10^{-3}).

Isokinetic Exercise. The external work recorded during the knee extension/flexion exercises was significantly greater than that recorded during the bouts of elbow extension/flexion exercise ($p<0.05$). The mean values for the knee extension/flexion exercises bouts ranged from 19.9 ± 2.5 $kJ \cdot min^{-1}$ at 0.52 $rad \cdot s^{-1}$, to 22.1 ± 3.6 $kJ \cdot min^{-1}$ at the 2.62 $rad \cdot s^{-1}$ velocity ($p>0.05$).

For the elbow extension/flexion exercises values ranged from 8.4 ± 2.6 $kJ \cdot min^{-1}$ at the 0.52 $rad \cdot s^{-1}$ velocity, to 9.0 ± 1.4 $kJ \cdot min^{-1}$ at the 1.57 $rad \cdot s^{-1}$ velocity ($p>0.05$).

In response to both knee and elbow extension/flexion exercise bouts, HR increased significantly ($p<0.01$). The HR response to the knee extension/flexion exercises was greater than that noted for the elbow extension/flexion exercise at 0.52 $rad \cdot s^{-1}$ ($p<0.05$). However, for a given exercise there were no significant differences between the three velocities. The SV recorded during the isokinetic exercise bouts was not significantly different from the pre-exercise value. As a result of the changes in HR, CO increased significantly, for both the knee extension/flexion and the elbow extension flexion exercises ($p<0.01$). For a given exercise velocity, the CO response to the bouts of knee extension/flexion exercise tended to be larger than that observed during the elbow extension/flexion exercises ($p<0.05$). These findings are summarized in Table 3.1.

The MAP rose significantly during all isokinetic exercise bouts ($p<0.01$). The highest MAP was recorded during the knee extension/flexion exercises ($p<0.05$). For a given exercise (knee or elbow extension/flexion) the magnitude of the increase in MAP was similar for the three velocities (Table 3.1). For both the knee and elbow extension/flexion exercises a significant reduction in SVR was recorded, as compared to the pre-exercise values ($p<0.05$). The increase in RPP recorded during knee extension/flexion exercises was significantly higher than that observed during the bouts of elbow extension/flexion exercise ($p<0.05$).

Isometric Exercise. The HR response to isometric contraction of the leg was lower than that recorded during the isokinetic knee extension/flexion exercise ($p<0.05$). For the isometric contraction about the leg the SV response was significantly lower than the pre exercise value ($p<0.05$). The SV response to the isometric contraction of the leg was also significantly lower than that recorded during the isokinetic knee extension/flexion exercises ($p<0.05$). The isometric contraction of the arm was not associated with a significant reduction in SV, compared to either the pre exercise value or that recorded during isokinetic elbow extension/flexion. The CO response to the isometric contraction of the leg was lower than that recorded during isokinetic knee extension/flexion exercise ($p<0.05$). The increase in CO during the isometric contraction of the arm was significantly lower than that observed during elbow extension/flexion exercise at 1.57 or 2.62 $rad \cdot s^{-1}$ ($p<0.05$).

The MAP recorded during the isometric contraction of the leg was significantly higher than that recorded during the isometric contraction of the arm ($p<0.05$). When the isometric and isokinetic exercise bouts were compared, the MAP recorded during the isometric contraction of the leg was significantly higher than that recorded during the bouts of isokinetic knee extension/flexion exercise ($p<0.05$). The SVR calculated for the isometric contractions was not changed from the pre-exercise level. The RPP calculated for the isometric contraction of the leg was significantly greater than the RPP calculated for the knee extension/flexion exercises at 0.52 and

1.57 rad$\cdot$s^{-1} ($p<0.05$).

Discussion

The primary objective of this study was to define the acute cardiovascular response to isokinetic exercise. To achieve this aim healthy young male subjects performed maximal knee extension/flexion and elbow extension/flexion exercises at each of 3 velocities. The study design also allowed for a comparison of these cardiovascular responses with those which occur during maximal isometric and dynamic exercise.

The cardiovascular responses to maximal upright exercise on a bicycle ergometer in the "steady state" are well known (1,21,22). Dynamic exercise is accompanied by relatively linear increases in HR and MAP. The SV increases linearly from rest until the HR exceeds 110-120 beat$\cdot$min^{-1} (6). At higher heart rates the SV remains unchanged until maximum exertion is achieved, at which time there may be a small decrease (6,23). Thus, at relatively low levels of exercise the CO is increased by a combination of an enhanced SV and HR. At high levels of exertion, the HR alone appears to play a major role in generating the increases in CO (1). However, it has to be recognized that this apparent constancy of the SV is maintained by an increase in contractility of the cardiac muscle (11,24). These changes are accompanied by a reduction in the overall SVR. In the present study, these adaptations were evident during upright cycle ergometry.

The cardiovascular responses to maximal isokinetic exercise were different from those observed during maximal dynamic exercise. The nature of the protocol was such that the measurements were not made during "steady state"; each exercise bout being one minute in duration. During isokinetic exercise the increase in CO was due primarily to the changes in HR. These changes were accompanied by an increase in MAP and a reduction in SVR. Both HR and MAP increased at the onset of exercise and remained elevated for the duration of the isokinetic exercise bout. The rapid HR response was likely to be due to vagal withdrawal (21). The changes observed in HR and MAP during the elbow extension/flexion exercises, were qualitatively similar to those seen during the knee extension/flexion exercises, but the absolute values achieved were smaller. For the three velocities examined the HR and MAP responses (knee extension/flexion or elbow extension/flexion) were similar. These two features suggest that the magnitude of the HR and MAP responses may be a function of the active muscle mass and independent of the velocity of the exercise.

Mitchell et al (13) has defined the exercise pressor response as all the cardiovascular changes that serve to increase MAP during muscular contraction. During exercise, the magnitude of the increase in HR and MAP has been related to the relative exercise intensity and the size of the active muscle mass (9,14). With a larger muscle mass or a higher relative exercise intensity, greater increases in HR and MAP could be anticipated as a result of a greater degree of excitation of muscle afferent receptors (14). Thus during isokinetic exercise, one might expect the magnitude of the pressor response to be correlated with the mass of the exercising muscle. In the present study the values for the change in HR and MAP did not appear to be related to the velocity of the movement. Yet, the knee extension/flexion exercises produced a greater response than the elbow extension/flexion exercise bouts. Therefore, it is suggested that isokinetic exercise, even at the slowest velocity used in this study, provided a maximal stimulus to the muscle afferents which mediated the reflex responses.

During exercise, CO is influenced by the venous return, the magnitude of which is determined by the mass of the active muscle pump. As a larger muscle mass was utilized during dynamic exercise, a greater muscle pump would have been involved. Thus it was not surprising that the change in SV and CO during isokinetic exercise was small, compared to that recorded at fatigue in the dynamic exercise. The observation that SV and CO were not influenced by the velocity of the isokinetic exercise suggests that the muscle pump was maximal at the three velocities examined.

A number of reports (10,19,20) have shown that the normal cardiovascular response to isometric exercise is an elevated MAP mediated primarily by an increased CO with minimal contribution by SVR (10). Our observations for static contraction of the arm and leg are consistent with this claim. However, the exercise mode apparently affected the manner in which the pressor response was achieved. The pressor response to the isometric exercises was mediated by an increase in HR, and therefore CO, without a significant change in SVR. During the isokinetic exercises the pressor response was produced by proportionately larger changes in

CO. The differences in SVR were likely to be caused by a mechanical hinderance to blood flow in the tonically contracted muscles during the isometric contraction and by metabolic vasodilation in the rhythmically working muscles of the isokinetic exercise (1,10,21).

There are certain practical implications in these data. First, maximal isokinetic exercise did not precipitate the extreme increases in MAP that have been reported during near maximal weight lifting (12). Second, the cardiovascular stress associated with maximal isokinetic exercise, (the increase in HR, MAP, and RPP) appear to be proportional to the active muscle mass. It is suggested that isokinetic exercise might be adaptable to selected cardiac patients, with a view to improving their muscular strength and endurance.

TABLE 3.1 Changes in cardiovascular parameters during maximal isokinetic and isometric exercise.

Velocity $(\mathrm{rad \cdot s^{-1}})$	HR $(\mathrm{b \cdot min^{-1}})$	SV $(\mathrm{ml \cdot beat^{-1}})$	CO $(\mathrm{l \cdot min^{-1}})$	MAP (mmHg)	SVR $(\mathrm{dyn \cdot s \cdot cm^{-5}})$	RPP (HR x SBP x 10^{-3})
Arm Exercises						
Pre Exercise	70 ± 7 a	115 ± 9 b	8.1 ± 0.7 a	91 ± 6 a	904 ± 64 cd	9.1 ± 0.6 a
Isokinetic						
0.52	107 ± 8 bc	106 ± 11 ab	11.3 ± 0.8 bc	112 ± 6 bc	793 ± 54 b	18.4 ± 0.9 b
1.57	113 ± 8 bcd	105 ± 10 ab	11.9 ± 0.5 c	115 ± 5 c	773 ± 44 ab	18.8 ± 1.0 b
2.62	112 ± 6 bcd	103 ± 9 ab	11.5 ± 0.6 c	115 ± 6 c	809 ± 64 bc	19.0 ± 0.8 b
Isometric	102 ± 4 b	98 ± 10 ab	10.0 ± 0.8 b	121 ± 5 cd	969 ± 74 d	20.1 ± 1.7 b
Knee Exercises						
Velocity $(\mathrm{rad \cdot s^{-1}})$	HR $(\mathrm{b \cdot min^{-1}})$	SV $(\mathrm{ml \cdot beat^{-1}})$	CO $(\mathrm{l \cdot min^{-1}})$	MAP (mmHg)	SVR $(\mathrm{dyn \cdot s \cdot cm^{-5}})$	RPP (HR x SBP x 10^{-3})
Pre Exercise	69 ± 4 a	108 ± 4 b	7.4 ± 0.4 a	102 ± 4 b	1088 ± 88 e	9.9 ± 0.7 a
Isokinetic						
0.52	126 ± 9 de	111 ± 10 b	14.0 ± 0.9 d	135 ± 7 e	771 ± 57 ab	25.4 ± 2.0 c
1.57	128 ± 7 e	107 ± 9 b	13.7 ± 1.0 d	128 ± 5 de	747 ± 70 bc	24.2 ± 2.5 c
2.62	136 ± 7 e	111 ± 11 b	15.1 ± 0.8 d	129 ± 4 de	683 ± 38 a	26.3 ± 1.6 cd
Isometric	116 ± 5 cd	92 ± 6 a	10.4 ± 0.8 b	150 ± 7 f	1154 ± 56 d	29.8 ± 2.3 d

Values are mean ± SEM; n = 5. HR = heart rate; SV = stroke volume; CO = cardiac output; MAP = mean arterial pressure; SVR = systemic vascular resistance; RPP = rate pressure product; Values with similar suffixes denotes no significant difference ($p > 0.05$).

Figure 3.1 Recording of peak torque heights during a isokinetic knee extension/flexion exercise with maximal effort. The initial movement is extension followed by flexion. The exercise velocity in this example was constant at 1.57 rad·s^{-1}. The torque scale was set at a maximum of 244 N·m. and was recorded at a paper speed of 5 mm·s^{-1}.

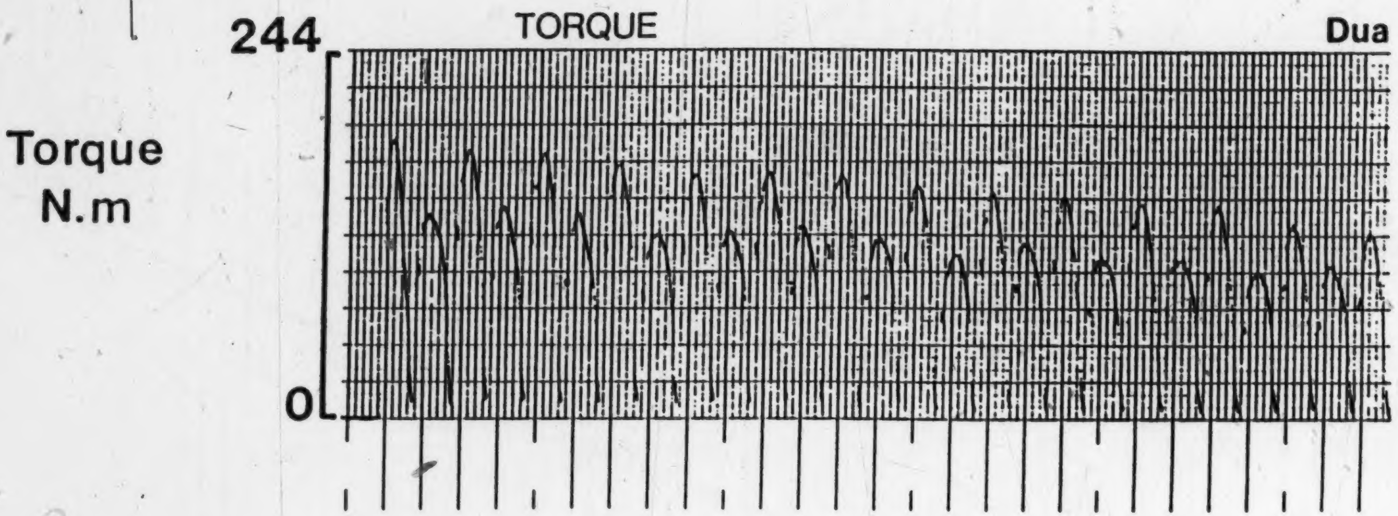
Torque
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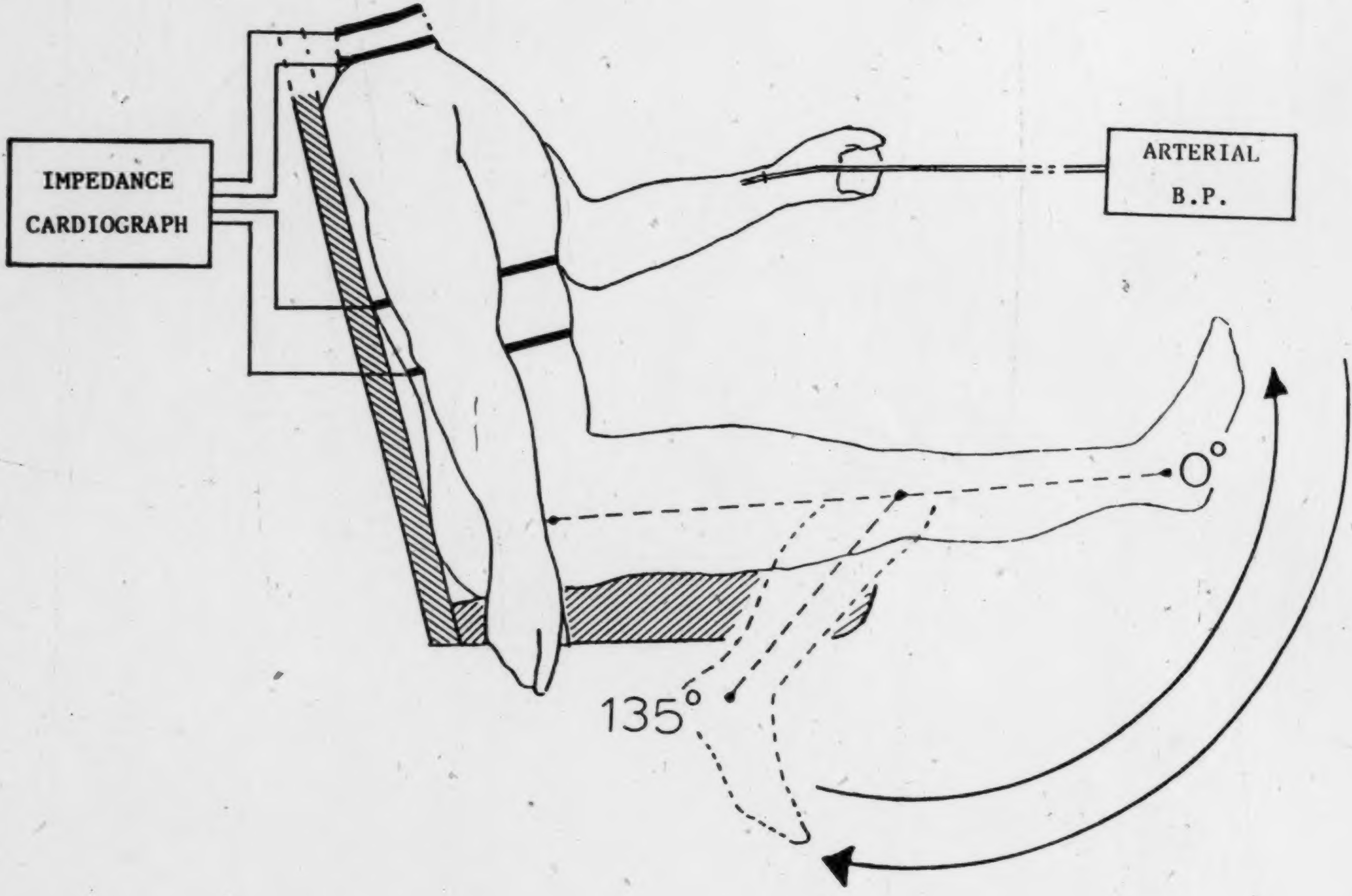
Figure 3.2 Schematic representation of the subject positioned for the knee extension/flexion exercise on the Cybex table. The figure also shows the relative positioning of the subject the recording monitors for impedance cardiography and the measurement of inter-atrerial blood pressure.


IMPEDANCE
CARDIOGRAPH
ARTERIAL
B.P.
0°
135°

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CHAPTER 4

EFFECTS OF HYDRAULIC CIRCUIT TRAINING ON CARDIOVASCULAR FUNCTION*

Introduction

Circuit weight training is a form of strength training in which a series of exercises are performed with resistance equipment in a predetermined sequence. In most circuit weight training programs, 8 to 12 different exercises are performed, usually at an intensity equivalent to 40 to 60% of the maximal force generating capacity of the participating muscles (10). Short work bouts, which incorporate 10 to 20 repetitions of each exercise are alternated with relief intervals during which time the individual moves from one station to another. While there is little doubt that circuit weight training improves muscular strength and endurance (2,11,12,29), the cardiovascular training response remains controversial. Several investigators (9,29) have reported small but significant improvements in maximal aerobic power with circuit weight training. However, others (2,18) found no improvement in aerobic power with such training. These findings are based upon exercise tests undertaken on bicycle ergometers (2) and treadmills (9,18,29). Further, it has been reported that the hemodynamic response to submaximal exercise on a bicycle ergometer (2), or a treadmill (18) is unchanged following circuit weight training.

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The controversy regarding the efficacy of circuit weight training on cardiovascular fitness may be related to the relative intensity of exercise used in the programs (10,29). While the heart rate response to traditional circuit weight training is within the range recommended by the American College of Sports Medicine (3), the aerobic demand has been shown to be insufficient to enhance maximal aerobic power (16,18,30). Recently however, circuit training on devices which provide accommodating resistance from variable hydraulic cylinders has been reported to produce both the metabolic and the cardiovascular stress necessary for the enhancement of maximal aerobic power (19). These hydraulic devices allow high intensity concentric exercise for the agonist and antagonist muscle groups involved in a given movement (23). Data has been presented to suggest that use of such devices will result in improved muscular strength and endurance (31). It is not known however, whether circuit training using hydraulic devices will elicit an improvement in the maximal aerobic power of previously untrained middle-aged men.

The purpose of the present study was to define the changes in maximal aerobic power induced by hydraulic circuit training and to compare them with the effects induced by a conventional dynamic exercise training program.

Methods

1) Subject Selection

Thirty-two male volunteers participated in this study. The subjects were instructed as to the nature of the study and written informed consent was obtained. Prior to training a medical history, physical examination (Appendix C), resting ECG, and a Graded Exercise Test (GXT) on a bicycle ergometer were performed. None of the subjects had clinical evidence of heart disease. Twenty-four of the subjects were randomly assigned to one of three groups (n = 8 in each); a) a non-exercising control group; b) a high intensity circuit training group, which completed the maximum number of repetitions possible during each work interval (HCT_{max}); and c) a high intensity circuit training group which completed a 70-85% of the maximum number of repetitions possible during each work interval (HCT_{sub}). Their results were compared with the responses of eight men of similar age and physical characteristics who participated in a dynamic exercise training program.

2) Measurement of Maximal Aerobic Power

Maximal aerobic power was assessed by measuring the maximum oxygen uptake (VO_2 max) achieved during a GXT on a bicycle ergometer (model 740, Siemens Electric Ltd., Missassuaga Ontario). The initial workload was set at 20 watts (W). At 3-minute intervals it was increased successively to 30, 50, and 80 W, and in steps of 50 W thereafter. The GXT continued until one of the following end points was attained: a leveling off of, or decrease in VO_2, with increasing workloads; attainment of 95% age predicted maximal heart rate or; volitional exhaustion (Borg scale reading >18). The VO_2 was measured at each workload using a continuous flow technique (1). The highest VO_2 value obtained during the GXT was recorded as VO_2 max.

3) Measurement of Cardiac Output

Stroke volume (SV), cardiac output (CO) and heart rate (HR) were measured by impedence cardiography (Minnesota Impedance Cardiograph, model 304A, Surcom Inc., Minneapolis, Minnesota). The use of this technique has been validated in this laboratory for endurance trained young male subjects and cardiac patients, both at rest and during exercise (28).

Readings were made at rest and immediately (within 3s) upon completion of each workload during the GXT. During the measurements, subjects were required to hold their breath at normal end-expiration to avoid artifact due to respiratory movement. The average of five cardiac cycles was used in the calculation of SV.

Blood pressure was measured using a mercury sphygmomanometer during the second minute of each stage of the GXT. The following parameters were calculated from the primary measurements; mean arterial pressure (mmHg) was calculated as the diastolic blood pressure + 1/3 pulse pressure; systemic vascular resistance ($dyn \cdot s \cdot cm^{-5}$) was estimated by dividing mean arterial pressure (MAP) by CO and multiplying by 80 (15). Left ventricular stroke work ($g \cdot m$), which was used as an index of left ventricular function, was calculated by the modified formula: SV ($ml \cdot beat^{-1}$) x MAP (mmHg) x 0.0136 (15). The rate pressure product was calculated by multiplying

HR and the systolic blood pressure (RPP = HR x SBP x 10^{-2}).

4) Measurement of Muscular Strength and Endurance

A Cybex II Isokinetic dynamometer (Lumex, Inc., Ronokonkoma, New York) equipped with a two channel recorder was used to measure muscular strength and endurance. Measurements were made during a series of maximal right knee, and right elbow extension/flexion exercises. Before the assessment, the subjects were familarized with the system and the required limb actions. Limb alignment and stabilization procedures were standardized for each test. Muscular strength was defined as the peak torque (N·m) recorded during a given exercise. The assessment of muscular strength was completed at two velocities, 1.05 and 3.14 rad·s^{-1}. Muscular endurance was defined as the total work accummulated over three 20s bout of maximal exercise at 3.14 rad·s^{-1}. In all cases the 1.05 rad·s^{-1} test was conducted first, with a minimum 5 minute recovery interval before the 3.14 rad·s^{-1} test. In order to minimize artifactual torques or the torque "overshoot" the first extension and flexion torques from each series were omitted from the results (27). External work, expressed in kiloJoules (kJ), was estimated for the three 20s work bout at 3.14 rad·s^{-1} by calculating the average torque (the area under the torque curve, in N·m·s^{-1} divided by the contraction time, in s) and then multiplying it by the angular displacement (rads) (24).

5) Training program

All subjects assigned to training exercised 30 minutes per day, three times a week for eight weeks. The training sessions commenced with a three minute warm-up and concluded with a three minute cool-down period. The dynamic training group exercised on bicycle ergometers for 24 minutes at a workload which corresponded to 70-85% of their heart rate reserve (21).

Those subjects assigned to hydraulic circuit training exercised on eight stations of variable resistance hydraulic equipment (Hydra-Fitness Canada Ltd, Sherwood Park Alberta). The following movement patterns were employed: knee extension and flexion; hip extension and flexion; hip abduction and adduction; elbow extension and flexion; shoulder extension and flexion; and planter flexion. Over the two weeks immediately preceeding training the subjects assigned to hydraulic circuit training were familarized with the equipment and the training circuit. Stations were arranged so as to exercise the upper and lower body alternately whenever possible. The circuit consisted of three 20s work intervals at each station with a 1:2 work:relief ratio.

The subjects assigned to the hydraulic circuit training were randomly allocated to one of two groups. One hydraulic circuit training group (HCT_{max}) completed the maximum number of repetitions possible during each work interval. The initial cylinder settings of 1, 3, and 2 (for the three successive work intervals of a given exercise) were increased when the subjects reached a level of one repetition per second (23).

The second hydraulic circuit training group (HCT_{sub}) trained at a level which corresponded to 70-85% of the maximum repetitions possible for a given exercise and cylinder setting. For the HCT_{sub}

group the initial cylinder settings were such that the subjects completed between 12-16 repetitions, when the movement involved a range of motion of less than 2.62 rads; and 8-12 repetitions when the exercise involved a range of motion of greater than 2.62 rads. For this group the maximum repetitions for each 20s work interval was established prior to training, and was reassessed at the completion of weeks 3 and 6. The adjusted programs were used for the subsequent weeks of training.

6) Acute Response to Training Sessions

The HR and blood pressure responses to the various training regimens were monitored during each session. The HR was monitored with Sport Testers (models pe 2000, or pe 3000; Polar/electro, Kempele Finland). The blood pressure response to training was assessed using an electronic sphygmomanometer (Infrasonde, model D4000, Puritan-Bennett Canada Ltd. Vancouver British Columbia). Use of the electronic sphygmomanometer was validated at rest and during steady state exercise. Systemic error was assessed by comparing with simultaneous measurement of blood pressure using a mercury sphygmomanometer. A significant linear correlation was obtained (N=20, regression coefficient = 0.82 $p<0.01$) between blood pressure measurements made using the electronic sphygmomanometer and those using a mercury sphygmomanometer. The slope of the regression line and the intercept were not different from unity and zero respectively, indicating no significant systemic error in the electronicc sphygmomanometer. The reproducibility of the blood pressure response was assessed in 6 subjects during steady state exercise at a power output of 50 watts, on two seperate days. A significant correlation was obtained ($r=0.88$, $p<0.001$) between the two days.

For the cycle training group HR and blood pressure were measured during steady state exercise. For the hydraulic circuit training groups, the HR response was recorded immediately upon completion of each work interval. The blood pressure response was assessed during the bilateral knee extension/flexion exercise.

7) Statistical Analysis

The comparison of the pre and post training measurements was made using a 2 way analysis of variance (6). A probability level of $p<0.05$ was accepted as the minimum value for statistical significance between groups. For a given variable if a significant F value was obtained, a test of Least Significant Difference was performed to assess the significance of the specific differences among the mean values. The data presented are expressed as means ± standard error of mean (SEM).

Results

The study was completed on 32 middle-aged males. Mean values for their physical characteristics were: age 42.2 ± 2.1 yrs; height 178.6 ± 2.4 cm; weight 83.2 ± 4.2 kg. The VO_2 max for the group, assessed on a bicycle ergometer was 2.67 ± 0.10 $l \cdot min^{-1}$. The features of the individuals in each of the four groups are given in Table 4.1. There were no significant differences between the four group in any of these variables, nor were any significant changes in body mass observed over the course of the study.

Acute Cardiovascular Response To Training Sessions

During the exercise sessions the three training groups attained an mean HR of 84% of maximum (Table 4.2). The mean HR response to training for the HCT_{sub} group was significantly lower than that recorded for the other two training groups ($p<0.05$). The MAP reponse during the training sessions was significantly higher for the HCT_{max} than for either the cycle or HCT_{sub} training groups ($p<0.05$). The rate pressure product, calculated from the training HR and SBP data, was significantly lower for the HCT_{sub} group than for the other two training groups ($p<0.05$)

Cardiovascular Responses to Training

a) Rest. Following training the resting HR was reduced significantly in the HCT_{sub} group ($p<0.05$). Both the cycling and HCT_{sub} groups demonstrated a significant increase in SV ($p<0.05$). Following training MAP was unchanged for the three training groups (Table 4.3), while left ventricular stroke work was significantly increased in the cycle trained group ($p<0.05$).

b) Submaximal Exercise. The cardiovascular response to submaximal exercise was assessed at a workload of 130 W. Following training the cycling and HCT_{sub} groups demonstrated a significant reduction in HR ($p<0.05$). For these same groups, the reduction in HR was associated with an increase in SV ($p<0.05$). All three training groups demonstrated a significant reduction in the calculated rate pressure product ($p<0.05$). The cycle trained and HCT_{max} groups also demonstrated a significant reduction in MAP ($p<0.05$). There were no significant changes in systemic vascular resistance or left ventricular stroke work for either the control or the exercising groups following training (Table 4.4).

c) Maximal Exercise. Following training, VO_2 max (Table 4.5) was significantly increased for both hydraulic circuit training groups, and the cycle training group ($p<0.05$). The largest increment in maximal aerobic power was recorded by the cycle training group ($p<0.05$). At maximal exercise the HR did not change significantly for either the control group, or the training groups. The SV at maximal exercise was significantly increased for the three training groups ($p<0.05$). As a result of the increase in SV, the maximal CO was significantly increased for all three training groups ($p<0.05$). At maximal exercise there were no significant changes in systemic vascular resistance, mean arterial pressure, or the calculated rate pressure product. Following training, left ventricular stroke work increased in the cycling and HCT_{sub} groups ($p<0.05$). The effects of the 8 weeks of training on the selected physiological variables at maximal exercise is presented in Table 4.6.

Muscular Strength and Endurance.

a) Knee Extension/Flexion. Alterations in peak torque consequent to the training programs are presented in Table 4.7. For the hydraulic circuit training groups the peak torque recorded during knee extension and flexion, at both the 1.05 and 3.14 $rad \cdot s^{-1}$ velocities was significantly increased following training ($p<0.05$). The cycle trained group demonstrated an increase in peak torque only during knee extension at 1.05 $\text{rad}\cdot\text{s}^{-1}$ ($p<0.05$). There were no significant changes in peak torque for knee extension/flexion in the control group.

b) Elbow Extension/Flexion. Following training, the peak torque recorded during elbow extension at 3.14 $\text{rad}\cdot\text{s}^{-1}$ was significantly increased for the hydraulic circuit training groups ($p<0.05$). During elbow flexion, a significant increase in peak torque was recorded at both velocities in the hydraulic circuit training groups ($p<0.05$). No changes in peak torque for elbow extension/flexion exercise were observed in the control or cycle trained groups.

c) Work. The accummulated work (kJ) recorded over three 20s work bouts about the knee at 3.14 $\text{rad}\cdot\text{s}^{-1}$ was increased for the three training groups (Figure 4.1). The cycle trained group demonstrated a 16% increase ($p<0.05$). A significantly larger increment was observed in the hydraulic circuit training groups; the HCT_{sub} and HCT_{max} groups recorded a 46 and 45% increase respectively ($p<0.05$).

For the three work bouts about the elbow (Figure 4.2), both hydraulic circuit training groups demonstrated significant increments in accummulated work. The HCT_{sub} group recorded a 34% increase whereas the HCT_{max} group recorded a 20% increment ($p<0.05$). No significant changes were observed in either the control or cycle training group.

Discussion

The present study was undertaken to determine the efficacy of hydraulic circuit training on the maximal aerobic power of previously untrained middle-aged adult males. To achieve this objective the effects of hydraulic circuit training were compared with the hemodynamic changes associated with dynamic exercise training on a bicycle ergometer. The principle hemodynamic responses to hydraulic circuit training included significant increases in VO_2 max, maximal SV, and CO, with relative bradycardia at submaximal workloads.

VO_2max. The increase in VO_2max ($l \cdot min^{-1}$) for the hydraulic circuit training groups represented a 9 and 13% improvement for the HCT_{sub} and HCT_{max} groups respectively. These findings are consistent with the report of Katch et al (19), who suggested that the magnitude of the HR and oxygen demands associated with hydraulic resistance exercise per se was sufficient to promote improvements in aerobic power. These increases are in agreement with previous reports on circuit weight training (9,29). The discrepancy between present findings and earlier circuit weight training studies which failed to demonstrate changes in VO_2 max (2,18) with training may be attributed to differences in the initial fitness level of the participants, and the relative exercise intensities of the circuit training programs (29).

The increase in VO_2 max observed in the cycle training group was similar to that reported by other studies using dynamic exercise training (7,25), but greater than that demonstrated by the hydraulic circuit training groups. The differences in the magnitude of the increase in VO_2 max for the cycle trained group and the hydraulic circuit trained groups may be attributed to differences in the oxygen costs of the two exercise modes. The energy expenditure estimates for circuit weight training (16,30), and hydraulic circuit training (19) are lower than those calculated for dynamic exercise, which could partially explain the differences in aerobic changes. Further, at half of the stations in the hydraulic circuit training program a smaller muscle mass (arms) was involved. It has been shown that the oxygen cost of arm work is approximately 70% of leg work at comparable heart rates (7,26). Therefore, the overall stimulus for aerobic improvement may have less for hydraulic circuit training than for cycling.

The fact that the change in maximal aerobic power was similar for the two hydraulic circuit training groups may be attributed to the work accomplished per training session (13). While the force per contraction was likely greater for HCT_{max} group because of the "all out" effort associated with their program, in many instances the number of repetitions completed per set were greater for the HCT_{sub} group. Consequently, the accumulated work per session may have been similar for the two groups.

Possible Mechanisms. In terms of the Fick principle any change in VO_2 max may be attributed to changes in maximal CO and arteriovenous oxygen difference (a-vO_2 diff). Within skeletal muscle training may induce changes in capillarization and oxidative enzyme activity. While increased oxidative potential in muscle after cycle training is well established (4,26) the data from strength trained muscle are inconclusive (14,17,20). Thus, while adaptations to the oxidative potential of skeletal muscle may be anticipated in the cycle trained group, such an observation can not be extended to the hydraulic circuit training groups. Investigations into the effect of exercise training on capillary supply have shown an increase in capillary density with endurance training (5,26), and that the time course changes for capillarization are similar to those of VO_2 max (4). The effects of strength training on capillary density is a topic currently under investigation. Data has been presented to suggest that the capillary density may be increased following a program of strength training (22).

As maximal HR did not change over the course of training, the increase in maximal CO, observed in the training groups, may be attributed to the changes in SV (Figures 4.3 and 4.4). The increased SV observed in the cycle training group is in agreement with previous reports on dynamic training (7). The finding of an increase in maximal SV with hydraulic circuit training is consistent with the hypothesis presented by Gettman et al (9). They reported an increase in O_2 pulse from which they inferred an increase in SV following a program of circuit weight training.

The changes in SV probably resulted from cardiac dimensional changes and extramyocardial adaptations. Changes in VO_2 max are parelleled by small but significant increases in total blood volume (7). Blomqvist and Saltin (7) noted a relationship between maximal stroke volume prior to volume loading and the magnitude of the increase in stroke volume after loading. They suggest that training alters the ventricular compliance characteristics by modifying left/right ventricular pericardium interaction, resulting in an increased diastolic reserve capacity. The finding of an increased left ventricular stroke work for the cycling and HCT_{sub} groups, at maximal exertion is consistent with an improved left ventricular function following training.

For the HCT_{sub} groups, the training-induced increase in SV was associated with a resting bradycardia (Table 4.3), and a decreased HR response to a given absolute workload. The resting bradycardia, following training may be attributed to increased parasympathetic tone (25). Both increased parasympathetic tone and a reduction in sympathetic stimulation account for the reduced HR response to a given absolute submaximal workload following training. The reduced sympathetic stimulation to a given absolute submaximal workload may be attributed to reduced peripheral afferent activity following training (7).

Muscular Strength and Endurance. Compared to the control and cycle trained groups, the hydraulic circuit training groups demonstrated significant increases in peak torque and accumulated work during knee extension/flexion and elbow extension/flexion exercises. The gains in muscular strength and endurance observed in the hydraulic circuit training groups were similar to those reported for other circuit weight training studies (9,18,29). The increases in strength and endurance can be explained on the basis of structural, neural, and biochemical adaptations (8,10,22,26). The fact that the increases were similar for the two hydraulic circuit training groups, suggesting that the HCT_{sub} training protocol was effective in improving muscular strength and endurance.

Summary

The present study indicates that hydraulic circuit training is effective in improving the maximal aerobic power, as well as the muscular strength and endurance of previously untrained middle-aged men. The improvements demonstrated by the HCT_{sub} group, at a reduced central circulatory demand (i.e. heart rate and rate pressure product), suggests that such exercise might be useful in training cardiac patients with depressed ventricular function. By training the various muscle groups one at a time, these patients may be able to enhance their aerobic power, muscular strength, and endurance at a reduced central hemodynamic load. Whether such training would be detrimental to such patients is not known and further studies on patients undergoing this form of exercise training is indicated.

Table 4.1 Pre training physical characteristics of the four groups.

	Control	Cycle	HCT_{sub}	HCT_{max}
AGE (years)	42.3 ± 3.1	41.9 ± 2.2	42.0 ± 1.8	41.9 ± 3.2
Height (cm)	179.4 ± 3.3	182.8 ± 2.1	177.4 ± 2.5	178.7 ± 2.0
Weight (kg)	82.3 ± 4.5	84.8 ± 2.7	80.9 ± 3.2	85.0 ± 3.4

Values are expressed as means ± SEM. N - 8 per group.

Table 4.2 Acute cardiovascular response to the training sessions.

	Cycle	HCT_{sub}	HCT_{max}
HR ($b \cdot min^{-1}$) (% of max)	149 ± 4 b (89%)	134 ± 3 a (78%)	143 ± 4 b (85%)
MAP (mmHg) (% of max)	100 ± 2 a (86%)	100 ± 1 a (89%)	107 ± 2 b (88%)
RPP (HR x SBP x 10^{-2}) (% of max)	228 ± 14 b (69%)	197 ± 6 a (61%)	220 ± 8 b (65%)

Values are expressed as means ± SEM. HR - heart rate; MAP - mean arterial pressure; RPP - rate pressure product. Values with similar suffixes denotes no significant difference ($p>0.05$). % of max = percentage of maximum value demonstrated at the pre-training graded exercise test.

Table 4.3 The cardiovascular parameters at rest.

	Control		Cycle		HCT_{sub}		HCT_{max}	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
HR	76 ±4	70 ±2	70 ±4	65 ±4	81 ±5	71 * ±4	72 ±2	71 ±4
SV	68 ±4	71 ±4	77 ±5	91 * ±6	64 ±5	75 * ±4	72 ±3	77 ±5
CO	5.1 ±0.2	5.0 ±0.3	5.3 ±0.2	5.9 ±0.5	5.1 ±0.4	5.2 ±0.2	5.2 ±0.3	5.3 ±0.3
MAP	91 ±2	84 * ±3	88 ±1	88 ±3	85 ±2	85 ±2	91 ±3	88 ±2
SW	84 ±6	81 ±6	93 ±6	108 * ±5	75 ±6	86 ±5	90 ±5	91 ±5

Values are expressed as means ± SEM. HR - heart rate (b·min^{-1}); SV - stroke volume (ml·beat^{-1}); CO - cardiac output (l·min^{-1}); MAP - mean arterial pressure (mmHg); SW - stroke work (g·m).

* - pre vs post data significantly different ($p<0.05$).

Table 4.4 The cardiovascular response at a workload of 130 watts on a bicycle ergometer.

	Control		Cycle		HCT_{sub}		HCT_{max}	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
VO2	1.82 ±0.04	1.80 ±0.05	1.86 ±0.02	1.87 ±0.02	1.83 ±0.03	1.80 ±0.04	1.83 ±0.03	1.87 ±0.03
HR	134 ±6	135 ±6	122 ±5	110 * ±3	138 ±4	123 * ±4	133 ±2	125 ±3
SV	100 ±4	99 ±4	110 ±5	122 * ±4	100 ±4	111 * ±3	103 ±3	110 ±4
CO	13.2 ±0.1	13.2 ±0.2	13.3 ±0.1	13.4 ±0.2	13.6 ±0.2	13.6 ±0.7	13.6 ±0.2	13.7 ±0.3
MAP	105 ±2	99 ±2	101 ±3	92 * ±2	100 ±2	94 ±2	111 ±3	104 * ±2
SVR	634 ±12	600 ±11	608 ±17	554 ±17	587 ±14	553 ±22	651 ±19	618 ±13
RPP	217 ±12	211 ±11	193 ±11	155 * ±6	221 ±10	183 * ±15	224 ± 7	205 * ±6
SW	143 ±6	133 * ±6	152 ±7	153 ±5	135 ±5	142 ±4	155 ±7	155 ±4

Values are expressed as means ± SEM. HR - heart rate ($b \cdot min^{-1}$); SV - stroke volume ($ml \cdot beat^{-1}$); CO - cardiac output ($l \cdot min^{-1}$); MAP - mean arterial pressure (mmHg); SVR - systemic vascular resistance ($dyn \cdot s \cdot cm^{-5}$). RPP - rate pressure product (HR x SBP x 10^{-2}); SW - stroke work ($g \cdot m$). * - pre vs post training data significantly different ($p<0.05$).

Table 4.5. Changes in VO_2 max expressed in absolute and relative terms over the eight week study period.

Group	VO_2 max ($l \cdot min^{-1}$)		VO_2 max ($ml \cdot kg^{-1} \cdot min^{-1}$)	
	Pre	Post	Pre	Post
Control	2.52 ± 0.09	2.47 ± 0.10	30.7 ± 2.1	30.0 ± 2.2
Cycle	2.80 ± 0.09	3.22 ± 0.10 *	33.0 ± 2.5	38.3 ± 1.1 *
HCT_{sub}	2.68 ± 0.10	2.91 ± 0.11 *	33.1 ± 1.1	35.9 ± 1.3 *
HCT_{max}	2.66 ± 0.08	3.01 ± 0.15 *	31.3 ± 1.6	35.6 ± 1.4 *

Values are mean ± SEM, n = 8 per group. * pre vs post data significantly different ($p<0.05$).

Table 4.6 Cardiovascular response maximal exercise on a bicycle ergometer.

	Control		Cycle		HCT_{sub}		HCT_{max}	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
HR	167 ±5	167 ±4	172 ±4	168 ±4	171 ±5	167 ±4	167 ±2	168 ±1
SV	100 ±5	99 ±4	109 ±4	123 * ±4	100 ±4	112 * ±4	102 ±2	110 * ±2
CO	16.6 ±0.7	16.4 ±0.7	18.6 ±0.6	20.6 * ±0.5	17.1 ±0.7	18.7 * ±0.7	17.1 ±0.3	18.5 * ±0.4
MAP	115 ±2	109 ±3	116 ±3	112 ±2	112 ±3	110 ±2	121 ±4	117 ±2
SVR	558 ±19	536 ±25	502 ±21	440 ±14	526 ±21	477 ±22	568 ±17	513 ±12
RPP	322 ±11	306 ±10	336 ±10	319 ±12	325 ±14	319 ±15	341 ±5	336 ±10
SW	156 ±8	146 ±7	171 ±7	189 * ±7	152 ±6	166 * ±4	170 ±8	177 ±5

Values are expressed as means ± SEM. HR - heart rate (b·min^{-1}); SV - stroke volume (ml·beat^{-1}); CO - cardiac output (l·min^{-1}); MAP - mean arterial pressure (mmHg); SVR - systemic vascular resistance (dyn·s·cm^{-5}); RPP - rate pressure product (HR x SBP x 10^{-2}); SW - stroke work (g·m). * - pre vs post training data significantly different ($p<0.05$).

Table 4.7 Changes in Peak torque (N·m) following training.

	Angular Velocity (rad·s^{-1})							
	Extension				Flexion			
	1.05		3.14		1.05		3.14	
Knee	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Control	199 ±14	190 ±16	101 ±3	104 ±6	104 ±5	99 ±4	75 ±3	80 ±4
Cycle	188 ±5	200 * ±6	110 ±6	110 ±4	111 ±8	121 ±6	78 ±6	82 ±3
HCT_{sub}	176 ±11	216 * ±8	100 ±8	130 * ±4	103 ±4	127 * ±6	71 ±5	87 * ±7
HCT_{max}	187 ±12	209 * ±9	105 ±4	137 * ±4	100 ±5	119 * ±4	72 ±4	91 * ±3
Elbow								
Control	53 ±3	54 ±2	31 ±2	32 ±2	44 ±3	45 ±3	30 ±2	29 ±1
Cycle	53 ±2	55 ±2	32 ±1	36 ±1	53 ±2	50 ±3	34 ±1	33 ±2
HCT_{sub}	53 ±4	59 ±5	32 ±4	40 * ±2	54 ±3	62 * ±3	29 ±4	37 * ±2
HCT_{max}	50 ±3	56 ±5	30 ±2	37 * ±3	44 ±2	60 * ±3	30 ±2	38 * ±1

Values are group means ± SEM expressed in N·m. * - pre vs post data significantly different ($p<0.05$).

Figure 4.1. Accummulated work over three 20s work bout of knee extension/flexion exercise at 3.14 rad·s^{-1}.

* - pre vs post significantly different ($p<0.05$).

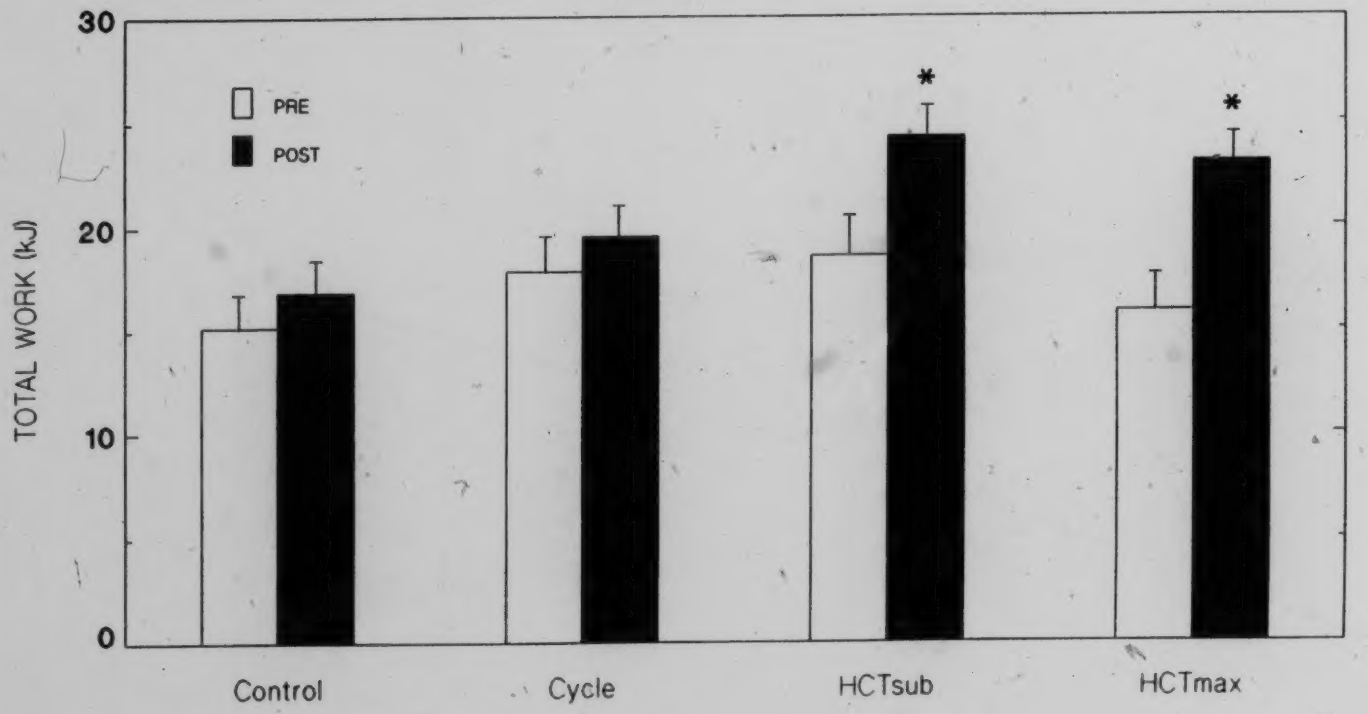

30
20
10
0
TOTAL WORK (kJ)
PRE
POST
*
*
Control
Cycle
HCTsub
HCTmax

Figure 4.2. Accummulated work over three 20s work bout of elbow extension/flexion at 3.14 rad·s^{-1}. * - pre vs post significantly different ($p<0.05$).

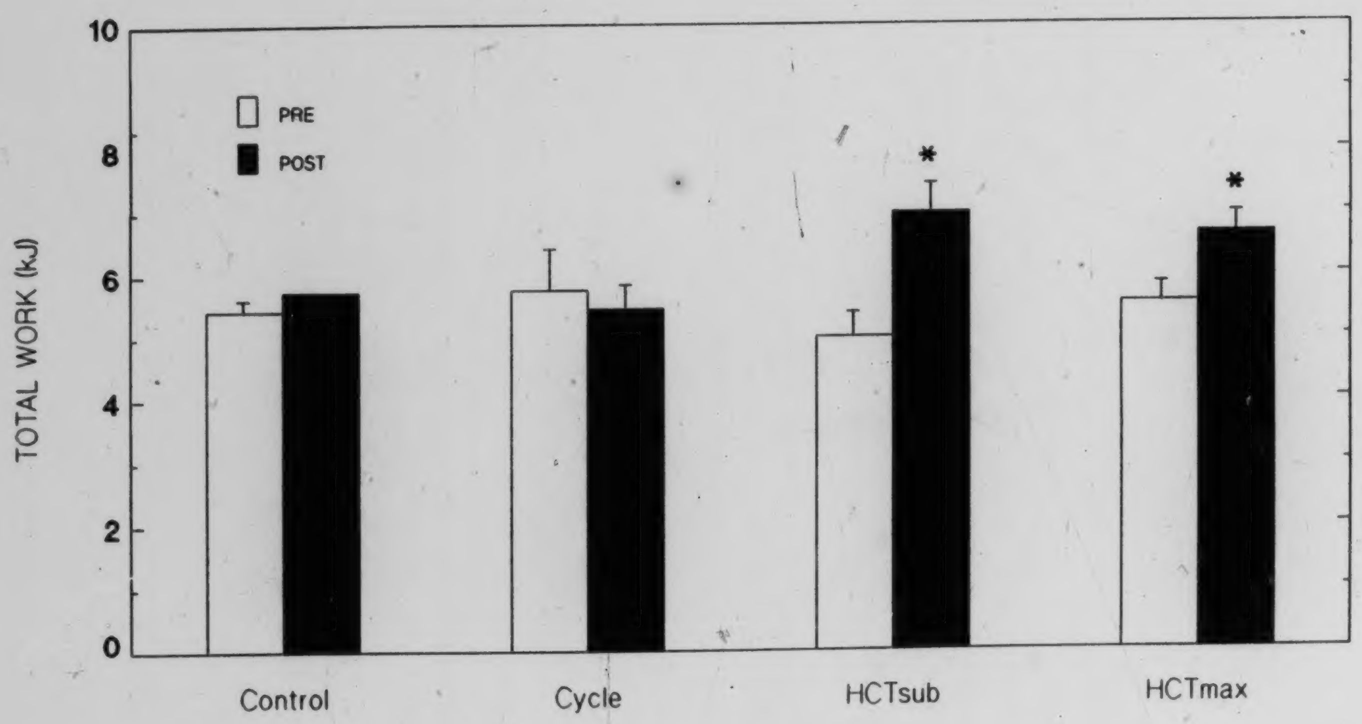

10
8
6
4
2
0
TOTAL WORK (kJ)
PRE
POST
*
*
Control
Cycle
HCTsub
HCTmax

Figure 4.3. Changes in the stroke volume response to exercise in the Control (A) and Cycle (B) trained groups. * - pre vs post data significantly different (p<0.05).

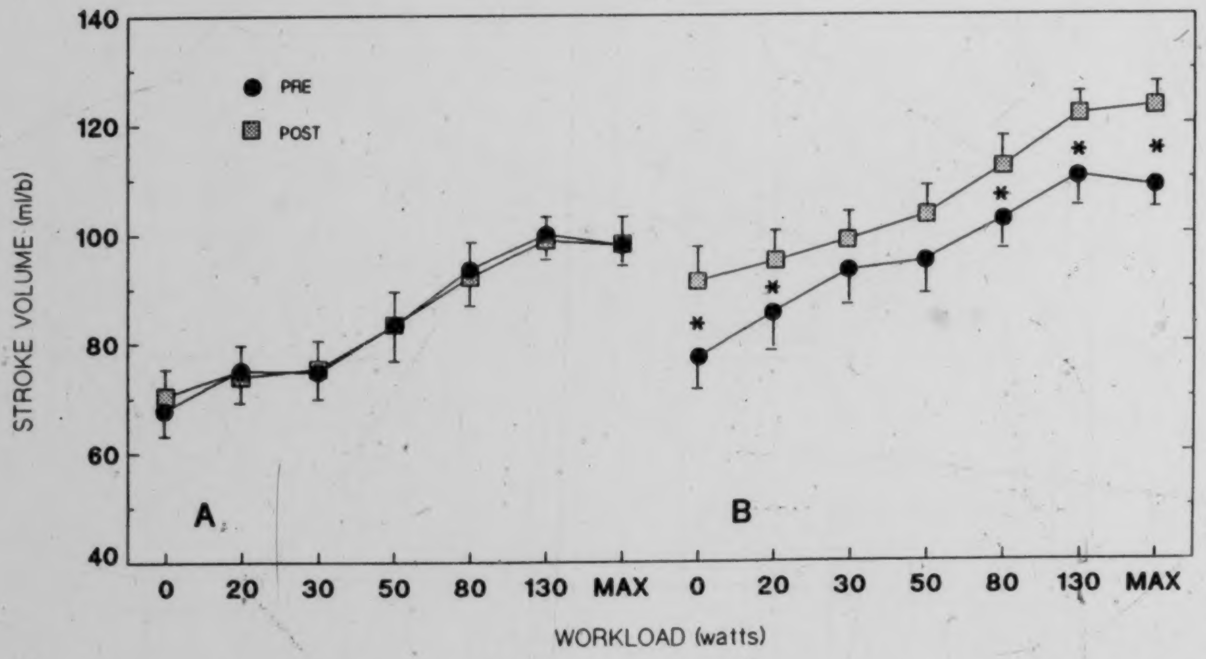
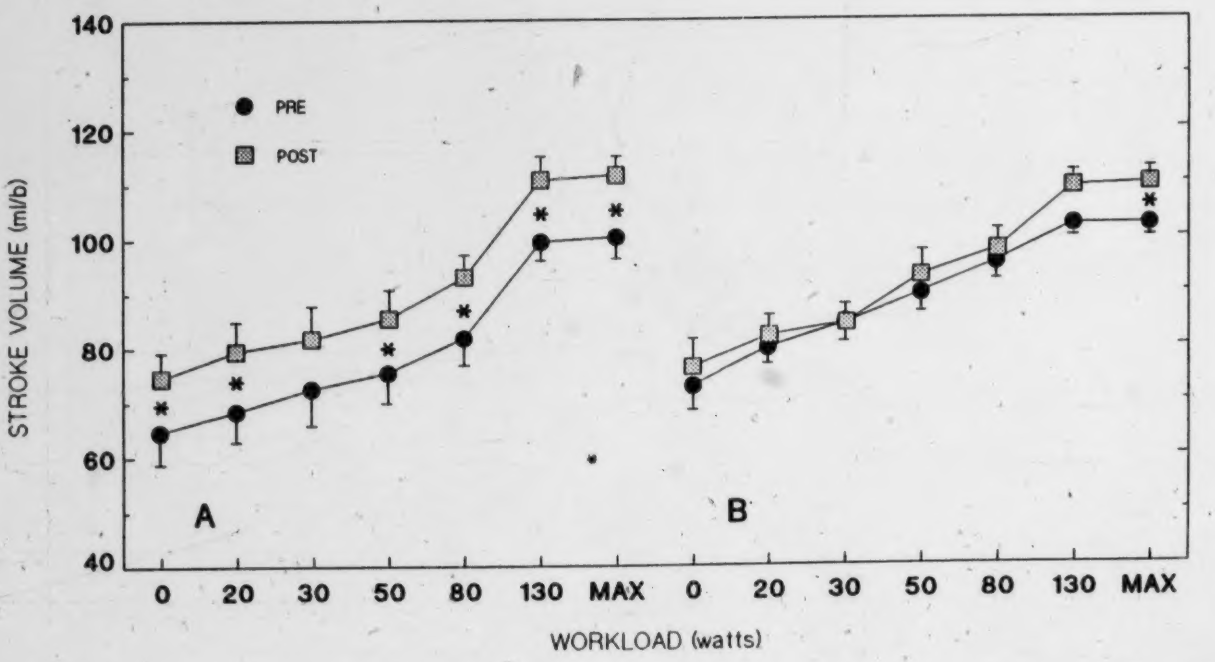

PRE
POST
STROKE VOLUME (ml/b)
140
120
100
80
60
40
A
B
*
0
20
30
50
80
130
MAX
0
20
30
50
80
130
MAX
WORKLOAD (watts)

Figure 4.4. Changes in the stroke volume response to exercise in the HCT_{sub} (A) and HCT_{max} (B) trained groups. * - pre vs post data significantly different ($p<0.05$).



● PRE
▨ POST
STROKE VOLUME (ml/b)
140
120
100
80
60
40
A
B
*
0
20
30
50
80
130
MAX
0
20
30
50
80
130
MAX
WORKLOAD (watts)

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CHAPTER 5

HYDRAULIC CIRCUIT TRAINING FOLLOWING CORONARY ARTERY BYPASS SURGERY*

Introduction

Aerobic exercise training has become an integral part of cardiac rehabilitation over the past thirty years. While there is little doubt that aerobic exercise training will enhance systemic oxygen uptake (6), the nature of occupational stresses, and the absence of a significant cardiovascular crossover effect with aerobic training calls into question its practical benefit. For instance, the physical demands of vocational energy expenditure are usually low, involving submaximal muscular efforts utilizing the upper extremeties (10). Few occupations require sustained walking or jogging (10). Thus, factors other than aerobic energy expenditure constitute the major physical stresses in daily living.

Given that improved exercise tolerance is the practical foundation of exercise rehabilitation programs an examination of alternate training modes has merit for cardiac patients who want to return to occupations requiring moderate levels of manual labor, or frequent lifiting.

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Circuit training is a form of strength training in which a series of exercises are performed with resistance devices in a predetermined sequence. Short work bouts, which incorporate 10 to 20 repetitions of each exercise are alternated with relief intervals. There are several practical advantages to hydraulic circuit training for cardiac patients (22). First, the equipment is capable of low power outputs with smaller muscle groups which should limit the demand for an increase in myocardial oxygen uptake during the actual exercise. Second, hydraulic resistance devices work the agonist and antagonist muscle groups concurrently and accommodates to the force produced over the range of motion, thus optimizing both exercise time and intensity. Another feature of this equipment relates to exercise skill requirements. Hydraulic devices are passive, thus minimizing the risk of muscular and joint strain which can result from efforts to control an applied force (i.e. lifting and lowering weights). Finally, hydraulic circuit training is performed in an interval fashion, so as to attenuate the need for a sustained stress on the cardiovascular system (25)

Traditionally, strength training exercises have not been included in cardiac rehabilitation, because it was felt that such exercise produce ischemic changes, and increased myocardial irritability (3). However, more recent studies have demonstrated the relative safety of weight carrying (8,30) and circuit weight training exercises (19,27,33) among selected cardiac patients. The addition of hydraulic circuit training to medically supervised cardiac exercise programs may enhance the patients' ability to meet many of the physical demands associated with their daily activities. Data has been presented to suggest that hydraulic circuit training will result in improved muscular strength, endurance, and aerobic power of previously untrained middle-aged men (see Chapter 4). However, it is not known whether hydraulic circuit training will elicit improvements in cardiac rehabilitation patients.

The purpose of the present study was to define the changes in maximal aerobic power, induced by hydraulic circuit training, and to compare them with the effects induced by a conventional aerobic exercise training program in a group of patients recovering from coronary artery bypass surgery.

Methods

1) Subject Selection

Eighteen male patients recovering from coronary artery bypass surgery participated in this study. The patients were instructed as to the nature of the study and informed consent was obtained. Prior to training a medical history, physical examination (Appendix C), resting ECG, and a Symptom Limited Graded Exercise Test (SL-GXT) on a bicycle ergometer were performed. Twelve of the patients were randomly assigned to either a cycle or circuit training group. Their results were compared with the responses from six patients who volunteered to serve as a non-exercising control group.

2) Measurement of Maximal Aerobic Power

Maximal aerobic power was assessed by measuring the symptom-limited maximum oxygen uptake (SL-VO_2 max) achieved during a SL-GXT on a bicycle ergometer (model 740, Siemens Electric Ltd., Missassuaga Ontario). All patients were familar with the apparatus. The initial workload was set at 30 watts (W) and increased by 20 W every 3 minutes until one or more of the following end points was achieved: a leveling off of, or decrease in VO_2 with increasing workloads; attainment of 85% age predicted maximal heart rate; severe dyspnea; volitional exhaustion (Borg scale reading >18); chest pain; dizziness; electrocardiographic evidence of ischemia; or an abnormal blood pressure response (32). The VO_2 was measured at each workload using a continuous flow technique (1). The highest VO_2 value obtained during the SL-GXT was recorded as SL-VO_2 max.

3) Measurement of Cardiac Output

Stroke volume (SV), cardiac output (CO) and heart rate (HR) were measured by impedance cardiography (Minnesota Impedance Cardiograph, model 304A, Surcom Inc., Minneapolis, Minnesota). The use of this technique has been validated in this laboratory for endurance trained young male subjects and cardiac patients, both at rest and during exercise (31).

Readings were made at rest and immediately (within 3s) upon completion of each workload during the SL-GXT. During the measurements, subjects were required to hold their breath at normal end-expiration to avoid artifact due to respiratory movement. The average of five cardiac cycles was used in the calculation of SV.

Blood pressure was measured, using a mercury sphygmomanometer during the second minute of each stage of the SL-GXT. The following parameters were calculated from the primary measurements; mean arterial pressure (mmHg) was calculated as the diastolic blood pressure + 1/3 pulse pressure; systemic vascular resistance ($dyn \cdot s \cdot cm^{-5}$) was estimated by dividing mean arterial pressure (MAP) by CO and then multiplying by 80 (13). Left ventricular stroke work ($g \cdot m$), which was used as an index of left ventricular function, was calculated by the modified formula: SV ($ml \cdot beat^{-1}$) x MAP (mmHg) x 0.0136 (13). The rate pressure product was calculated by multiplying heart rate and the systolic blood pressure (HR x SBP x 10^{-2}).

4) Measurement of Muscular Strength and Endurance

A Cybex II Isokinetic dynamometer (Lumex, Inc., Ronokonkoma, New York) equipped with a two channel recorder was used to measure muscular strength and endurance. Measurements were made during a series of knee, and shoulder extension/flexion exercises. Prior to the assessment the subjects were familarized with the system and the required limb actions. Limb alignment and stabilization procedures were standardized for each test. In order to minimize artifactual torques or the torque "overshoot" the first extension and flexion torques from each series were omitted from the results, and an optimal damping setting of 2 on the Cybex II chart recorder was used (29). Muscular strength was defined as the peak torque ($N \cdot m$) recorded during a given exercise. The assessment of muscular strength was completed at two velocities, 1.05 and 3.14 $rad \cdot s^{-1}$.

In all cases, the 1.05 $rad \cdot s^{-1}$ test was conducted first, with a minimum 5 minute recovery interval before the 3.14 $rad \cdot s^{-1}$ test. Muscular endurance was defined as the total work (kJ) accummulated over three 20s bouts of exercise at 3.14 $rad \cdot s^{-1}$, and was estimated by calculating the average torque (the area under the torque curve, in $N \cdot m \cdot s^{-1}$ divided by the contraction time, in s) and then multiplying it by the angular displacement (rads) (28). Upon completion of a given exercise the recordings of the torque curves were examined to ensure that the subject had exerted a maximal effort, which is characterized by a gradual degredation of peak torque height with repeated contractions.

5) Training program

Upon completion of all preliminary assessments, the training subjects were randomly allocated to either a cycle training or circuit training program. The cycle training group exercised on bicycle ergometers for 27 minutes at a workload that corresponded to 70-85% of their heart rate reserve (17).

Those subjects assigned to circuit training exercised on a three stations hydraulic resistance device (Total Power unit; Hydra-Fitness Canada Ltd. Sherwood Park Alberta). The following movement patterns were employed: knee extension and flexion; elbow extension and flexion (chest exercise); shoulder extension and flexion. Stations were arranged so as to exercise the upper and lower body alternately. The circuit consisted of three 20s work intervals at each station with a 1:2 work:relief ratio. Subjects completed 3 circuits per day. The circuit training corresponded to 70-85% of the maximum repetitions possible for a given exercise and cylinder setting. The initial cylinder settings were such that the 70-85% of the maximum repetitions possible corresponded to 12-16 repetitions, for the chest and knee exercises, and 8-12 repetitions for the shoulder exercise. The maximum repetitions for each 20s work interval was established prior to training and was reassessed at the completion of weeks 3 and 6. The adjusted programs were used for the subsequent weeks of training.

6) Cardiovascular Response to Maximal Hydraulic Resistance Exercise

Over the two weeks immediately preceeding training, all subjects were familarized with the cycle and circuit training programs. Upon completion of the familiarization period the cardiovascular response maximal hydraulic resistance exercise was assessed. The exercise session consisted of three circuits. Each circuit was comprised of chest, knee, and shoulder exercises. The subjects completed the maximum number of repetitions possible during a given work interval. Each exercise incorporated three 20s work intervals, with a 1:2 work:relief ratio. Simultaneous measurements of VO_2, and CO, were recorded at the completion of the third work interval of each exercise. The blood pressure response was measured using a standard sphygmomanometer during completion of the bilateral knee extension/flexion exercises. The data presented represent the averaged values for each exercise over the three circuits.

7) Acute Response to Training Sessions

The HR and blood pressure responses to the various training regimens were monitored during each training session. The HR was monitored continuously using a CM_5 lead, and a single channel electrocardiogram (Critical Care model 128A, Kontron Medical Ltd. Zurich Switzerland). The blood pressure response to training was assessed using an electronic sphygmomanometer (Infrasonde, model D4000, Puritan-Bennett Canada Ltd., Vancouver British Columbia). For the cycle training group HR and blood pressure were measured during steady state exercise. For the circuit training groups, the HR response was recorded immediately upon completion of each work and relief interval. The blood pressure response was assessed during completion of the bilateral knee extension/flexion exercise.

8) Statistical Analysis

The comparison of the pre and post training measurements was made using a 2 way analysis of variance (4). A probability level of $p<0.05$ was accepted as the minimum value for statistical significance between groups. For a given variable if a significant F value was obtained, a test of Least Significant Difference was performed to assess the significance of the specific differences among the mean values. The data presented are expressed as means ± standard error of mean (SEM).

Results

The study was completed on 18 male patients recovering from coronary artery bypass surgery. Mean values for their physical characteristics were: age 52.0 ± 2.9 yrs; height 176.4 ± 2.3 cm; weight 77.9 ± 3.8 kg. The VO_2 max for the group, assessed on a bicycle ergometer was 1.61 ± 0.09 $l \cdot min^{-1}$. The features of the individuals in each of the three groups are given in Table 5.1. There were no significant changes in body mass, for the cycle or circuit training groups, over the course of the study.

Acute Response to Hydraulic Resistance Exercise

The cardiovascular response to maximal hydraulic resistance exercise was compared with the values observed at maximal exercise during the initial SL-GXT (Table 5.2). The mean VO_2, for maximal hydraulic resistance exercise, represented 60% of the SL-VO_2 max demonstrated at the pretraining SL-GXT. The corresponding HR response averaged 86% of the value achieved at maximal exertion during the SL-GXT ($p<0.05$). The CO response was significantly increased during the hydraulic resistance exercise ($p<0.05$), however, the values achieved during the chest and shoulder exercises were lower than the maximum CO observed during the SL-GXT ($p<0.05$).

The comparison of the bilateral knee extension/flexion exercise with the SL-GXT revealed that MAP, SVR, and SW responses were comparable, whereas the calculated rate pressure product was lower during the hydraulic resistance exercise ($p<0.05$).

Acute Cardiovascular Response To Training Sessions

During the exercise sessions the two training groups attained a mean HR of 85% of maximum.(Table 5.3). The HR and MAP response to training for the cycle and circuit training groups represented a similar percentage of the maximum values observed at the pre-training SL-GXT. The rate pressure product, calculated from the training HR and SBP data, also represented a similar percentage of the maximum values observed at the initial SL-GXT.

Cardiovascular Responses to Training

a) Rest. Following training the resting HR was significantly lower in both the cycle and circuit training groups ($p<0.05$). For the cycle trained group the reduction in HR was associated with a significant increase in SV ($p<0.05$). There were no significant changes in resting HR or SV for the control group. The effects of the training programs on selected cardiovascular parameters at rest are presented in Table 5.4.

b) Submaximal Exercise. The cardiovascular response to submaximal exercise was assessed at a power output of 50 W. Following training the two exercising groups demonstrated a significant reduction in HR, and a corresponding increase in SV ($p<0.05$). The training groups also demonstrated a significant reduction, and in the calculated rate pressure product ($p<0.05$). No significant changes were observed in any of the monitored cardiovascular parameters for the control group (Table 5.5).

c) Maximal Exercise. The VO_2 max (Table 5.6) was significantly increased for both training groups ($p<0.05$). The increment in VO_2 max was similar for the two training groups and was associated with an increase in the accumulated work at the post training SL-GXT ($p<0.05$). At maximal exercise the HR did not change significantly for the control or training groups. The maximal SV was significantly increased for both training groups ($p<0.05$). As a result of the increase in SV, the maximum CO was significantly increased for the training groups ($p<0.05$). The effects of the 8 weeks of training on the selected cardiovascular variables at maximal exercise is presented in Table 5.7.

Muscular Strength and Endurance.

a) Knee Extension/Flexion. Alterations in peak torque consequent to the training programs are presented in Table 5.8. For the circuit training group the peak torque recorded during knee extension/flexion at 3.14 $rad \cdot s^{-1}$, and knee flexion at both velocities (1.05 and 3.14 $rad \cdot s^{-1}$) was significantly increased following training ($p<0.05$). The cycle trained group demonstrated an increase in peak torque during knee flexion at 1.05 $rad \cdot s^{-1}$ ($p<0.05$). There were no significant changes in peak torque for knee extension or flexion in the control group over the study period.

b) Shoulder Extension/Flexion. Following training, both the cycle and circuit training groups demonstrated an increase in peak torque during shoulder extension at 1.05 $rad \cdot s^{-1}$ ($p<0.05$). At the faster velocity (3.14 $rad \cdot s^{-1}$) only the circuit training group recorded a significant increase in peak torque ($p<0.05$). A significant increase in peak torque during shoulder flexion was recorded in the circuit training group at THE 3.14 $rad \cdot s^{-1}$ velocity ($p<0.05$). No changes in peak torque for shoulder extension/flexion were observed in the control group.

c) Work. The accummulated work recorded over three 20s work bouts of knee extension/flexion, at 3.14 $rad \cdot s^{-1}$, was increased for the two training groups (Figure 5.1). The cycle training group demonstrated a 31% increase whereas the circuit training group demonstrated a 29% increase ($p<0.05$).

For the three work bouts about the shoulder (Figure 5.2), the cycle training group recorded a mean increase of 26% ($p<0.05$). The circuit training group demonstrated a significantly larger increment (45%) over the study period ($p<0.05$).

Discussion

The present study was undertaken to determine the efficacy of hydraulic circuit training on the muscular strength, endurance, and maximal aerobic power of patients recovering from coronary artery bypass surgery. To achieve this objective the effects of hydraulic circuit training were compared with the hemodynamic changes associated with dynamic exercise training on a bicycle ergometer. The responses to circuit training included a significant increases in maximal aerobic power, muscular strength, and muscular endurance. The principle hemodynamic responses to circuit training included an increase in maximal CO, and SV, as well as a relative bradycardia at rest, and during submaximal exercise.

Acute Response to Maximal Hydraulic Resistance Exercise. The relative VO_2 and HR responses to maximal hydraulic resistance exercise were similar to those reported by Katch et al (18), suggesting that the magnitude of the oxygen cost, and HR response to hydraulic resistance exercise is sufficient to promote improvements in maximal aerobic power in post coronary bypass surgery patients. The disproportionate increase in HR, relative to VO_2 (85% vs 60% respectively) may be attributed to an increased sympathetic stimulation secondary to elevated plasma catecholamine levels during upper extremity strength training exercises (6,16). The increase in

SV during the hydraulic resistance exercises resulted from an increase in the relative preload, and myocardial contractility (5). The HR, CO, and rate pressure product responses, to maximal hydraulic resistance exercise, were all lower than the maximal values achieved during the SL-GXT. Present findings suggest that by stressing smaller muscle groups, one at a time for relatively short periods, this form of exercise did not precipitate the sustained heavy demands on the central circulation that were seen during maximal exercise on a bicycle ergometer.

Submaximal Exercise. It is generally recognized that aerobic training produces certain well defined changes considered to signify a gain in cardiovascular fitness. The best known manifestations include a reduction in both HR and systolic blood pressure at submaximal levels of exertion (6,11,14). Thus, the same degree of external work can be undertaken by these patients while generating a lower rate pressure product. After a period of training patients are able to achieve a greater work capacity while attaining the same rate pressure product as before training (6,14). The data obtained in the present study are in agreement with these general trends. These findings indicate that hydraulic circuit training is as effective as cycle training in enhancing cardiovascular fitness in patients recovering from coronary artery bypass surgery.

VO_2max. The studies completed to date have focused on the adaptations of post coronary artery bypass patients to aerobic training programs. The 18% increase in VO_2 max observed in the cycle training group was similar to that reported for other surgically treated patients following cycle training (14). The finding of an 18% increase in VO_2 max ($l \cdot min^{-1}$) for the circuit training group has not been reported previously. This finding is, however, consistent with results reported by Kelemen et al (19), who noted a 12% increase in treadmill time among cardiac patients who completed a 10 week circuit weight training program. Present findings are also in agreement with previous reports in healthy individuals (see Chapter 5;12,34).

The similarity in the magnitude of the increase in VO_2 max for the cycle and the circuit training groups was not anticipated. Previous studies (16,18) have suggested that the energy expenditure associated with circuit weight training (16) or hydraulic resistance exercise (18) is lower than that calculated for dynamic exercise. Indeed our assessment of hydraulic resistance exercise in post coronary artery bypass patients (Table 5.2) is consistant with this view. Therefore the overall stimulus for aerobic improvement in the circuit training group should have been lower. The fact that the cycle and circuit trained patients improved to a similar degree suggests that, for patients with low aerobic capacity, activities with lower oxygen costs may be incorporated into an exercise program designed to enhance aerobic power (6).

While the HR, MAP, and rate pressure product responses to the cycling and circuit training were similar (Table 5.3) it is important to note that, during the hydraulic circuit training, this level of cardiovascular stress was maintained for only 20s periods, which compares favorably with the sustained stress associated with the cycle training program. The implication of these findings is that improvement in maximal aerobic power in post coronary artery bypass patients can be achieved with short repeated bouts of exercise involving relatively small muscle groups.

Possible Mechanisms. Any increase in VO_2 max seen as a result of training may be attributed to changes in maximal CO and arteriovenous oxygen difference (a-vO_2 diff). For most cardiac patients a widening of the a-vO_2 diff characterizes the adaptive response to aerobic training (6,9,24,26). The failure to observe a widening of the a-vO_2 difference in the present study may be attributed to the length of the training program (6,24). Further, data on changes in the oxidative potential of skeletal muscle, following a variety of strength training programs has been inconclusive (7,15,20,21).

As maximal HR did not change over the course of the study, the increase in maximal CO observed in the training groups may be attributed to an increase in maximal SV (Figures 5.3). The increase in SV observed in the cycle trained patients is in agreement with previous reports on dynamic training following myocardial infarction (7,13,24). This increase in SV is generally associated with relatively high intensity training programs, and attributed to increased diastolic filling (6) and improved myocardial contractility (13). The finding of an increase in SV with hydraulic circuit training is consistent with reports from healthy individuals (Chapter 4;12). These findings suggest that the fundamental response of patients recovering from coronary artery bypass surgery is similar to that of healthy individuals.

The training-induced increase in SV was associated with a resting bradycardia, as well as a decreased HR response to submaximal workloads (Figure 5.3). The resting bradycardia may be attibuted to increased vagal tone following training (5). The reduced HR response to the various levels of submaximal exercise was mediated by a combination of increased parasympathetic tone and decreased sympathetic activity (5). The decreased sympathetic activity at a given absolute submaximal workload may be attributed to reductions in central command and systemic metabolic demand following training (5).

Muscular Strength and Endurance. Compared to the control and cycle trained groups, the circuit training group demonstrated consistent increases in peak torque and accumulated work during both knee extension/flexion, and shoulder extension/flexion. The increases in muscular strength and endurance may be attributed to structural, biochemical, and neural adaptations (7,20,21). The gains in muscular strength and endurance observed for the circuit training group were similar to those reported for circuit training programs in cardiac patients (19), or healthy individuals (Chapter 4;12). Present findings suggest that patients recovering from coronary artery bypass surgery can safely participate in an hydaulic circuit training program with the expectation of improving the muscular strength and endurance.

Summary

The present study indicates that hydraulic circuit training is effective in improving maximal aerobic power of patients recovering from coronary artery bypass surgery. The fact that these adaptations resulted from short repeated bouts of exercise which did not produce a sustained heavy demand on the central circulation suggests that this form of exercise may be a viable exercise mode for patients with left ventricular dysfunction, or who can tolerate only a limited amount of cardiovascular stress. The addition of a hydraulic circuit training to medically supervised cardiac exercise programs should enhance the patients ability to meet many of the physical demands associated with their daily activities.

Table 5.1. Pre-training characteristics of the patients.

	Control	Cycle	Circuit
AGE (years)	53.3 ± 1.4	51.7 ± 4.3	51.0 ± 3.1
Height (m)	178.0 ± 2.5	175.5 ± 2.1	175.7 ± 2.4
Weight (kg)	82.9 ± 4.3	72.4 ± 3.3	78.5 ± 3.8
Previous M.I.	3	3	3
Beta-receptor blockers	2	1	2
Weeks post surgery (mean)	9	9	10

Values are expressed as means ± SEM. N = 6 per group.

Table 5.2 Cardiovascular response to maximal hydraulic resistance exercise.

	REST	SL-GXT	HYDRAULIC RESISTANCE EXERCISE		
			Chest	Knee	Shoulder
VO_2	0.34 a ±0.08	1.61 c ±0.05	0.92 b ±0.05	1.02 b ±0.05	0.97 b ±0.09
HR	82 a ±3	138 c ±4	115 b ±6	118 b ±5	122 b ±6
SV	70 a ±3	100 b ±4	91 b ±8	102 b ±7	96 b ±7
CO	5.8 a ±0.3	13.9 c ±1.0	10.5 b ±1.1	12.0 bc ±1.1	11.7 b ±1.2
MAP	89 a ±2	112 b ±3	-	107 b ±4	-
SVR	1294 a ±63	651 b ±29	-	717 b ±71	-
RPP	98 a ±5	236 c ±12	-	177 b ±10	-
SW	86 a ±4	156 b ±5	-	151 b ±8	-

N - 18. VO_2 - oxygen uptake ($l \cdot min^{-1}$); HR - heart rate ($b \cdot min^{-1}$); SV - stroke volume ($ml \cdot beat^{-1}$); CO - cardiac output ($l \cdot min^{-1}$); MAP - mean arterial pressure; SVR - systemic vascular resistance ($dyn \cdot s \cdot cm^{-5}$). RPP - rate pressure product (HR x SBP X 10^{-2}) SW - stroke work ($g \cdot m$). SL-GXT - symptom limited graded exercise test. Values with similar suffixes denote no significant difference ($p<0.05$).

Table 5.3 Acute cardiovascular response to the training sessions.

	Cycle	Circuit
HR ($b \cdot min^{-1}$)	131 ± 3	108 ± 3
(% of max)	(87%)	(82%)
MAP (mmHg)	99 ± 5	96 ± 2
(% of max)	(87%)	(87%)
RPP ($HR \times SBP \times 10^{-2}$)	199 ± 8	174 ± 7
(% of max)	(73%)	(80%)

% of max - percentage of maximum value demonstrated at the pre-training SL-GXT.

Table 5.4 Cardiovascular parameters at rest.

	Control		Cycle		Circuit	
	Pre	Post	Pre	Post	Pre	Post
HR	83 ±4	80 ±3	84 ±3	73 * ±2	78 ±5	67 * ±4
SV	61 ±3	65 ±5	74 ±3	88 * ±2	72 ±4	78 ±5
CO	5.0 ±0.4	5.1 ±0.4	6.2 ±0.2	6.4 ±0.2	5.6 ±0.5	5.2 ±0.4
MAP	89 ±2	86 ±3	88 ±4	86 ±4	92 ±3	88 ±3
RPP	97 ±9	88 ±7	97 ±6	86 ±5	98 ±8	77 ±7
SVR	1434 ±45	1380 ±91	1147 ±57	1083 ±58	1357 ±100	1396 ±93
SW	73 ±4	75 ±7	90 ±7	104 ±5	91 ±8	92 ±4

HR - heart rate ($b \cdot min^{-1}$); SV - stroke volume ($ml \cdot beat^{-1}$); CO - cardiac output ($l \cdot min^{-1}$); MAP - mean arterial pressure (mmHg); SVR - systemic vascular resistance ($dyn \cdot s \cdot cm^{-5}$). SW - stroke work ($g \cdot m$). * pre vs post data significantly different ($p<0.05$).

Table 5.5. Cardiovascular responses at a submaximal workload of 50W.

	Control		Cycle		Circuit	
	Pre	Post	Pre	Post	Pre	Post
VO_2	0.94 ±0.03	0.97 ±0.03	0.91 ±0.03	0.92 ±0.02	1.01 ±0.05	0.95 ±0.02
HR	105 ±4	101 ±3	101 ±1	91 * ±2	100 ±6	87 * ±4
SV	83 ±5	87 ±4	93 ±4	104 * ±2	91 ±4	100 * ±3
CO	8.7 ±0.3	8.7 ±0.2	9.4 ±0.3	9.5 ±0.3	9.1 ±0.7	8.8 ±0.5
MAP	99 ±4	96 ±3	100 ±4	91 ±3	101 ±4	95 ±4
RPP	149 ±10	138 ±8	150 ±6	119 * ±4	142 ±12	113 * ±8
SVR	923 ±59	885 ±41	849 ±31	768 ±34	918 ±73	877 ±47
SW	111 ± 5	113 ±2	127 ±9	129 ±7	125 ±8	130 ±6

VO_2 - oxygen uptake ($l \cdot min^{-1}$); HR - heart rate ($b \cdot min^{-1}$); SV - stroke volume ($ml \cdot beat^{-1}$); CO - cardiac output ($l \cdot min^{-1}$); MAP - mean arterial pressure (mmHg); SVR - systemic vascular resistance ($dyn \cdot s \cdot cm^{-5}$). RPP - rate pressure product ($HR \times SBP \times 10^{-2}$) SW - stroke work ($g \cdot m$); * pre vs post data significantly different ($p<0.05$).

Table 5.6 Changes in SL-VO_2 max over the study period.

	Control		Cycle		Circuit	
	Pre	Post	Pre	Post	Pre	Post
VO_2 max ($l \cdot min^{-1}$)	1.54 ±0.10	1 61 ±0.08	1.64 ±0.10	1 93 * ±0.11	1.61 ±0.07	1.90 ±0.10
VO_2 max ($ml\ kg\ min^{-1}$)	18.6 ±1.1	19.5 ±1.0	22.7 ±1.2	26.9 * ±1.6	20.8 ±1.4	24.6 * ±1.6
Total Time (min)	14.0 ±0.9	15.0 ±0.6	15.2 ±1.1	19.0 * ±1.6	14.8 ±0.9	18.8 * ±1.7
Total Work (W)	940 ±111	1060 ±77	1157 ±127	1552 * ±200	1047 ±112	1545 * ±251
Highest WL (W)	107 ±7	113 ±3	117 ±6	140 * ±9	109 ±7	133 * ±12
SL-GXT Terminated Due to:						
fatigue	4	4	1	1	3	3
VO_2/H.R.	1	1	4	4	2	3
Symptoms	1	1	1	1	1	-

* pre vs post data significantly different ($p<0.05$).

Table 5.7 Cardiovascular response to maximal exercise.

	Control		Cycle		Circuit	
	Pre	Post	Pre	Post	Pre	Post
HR	132 ±5	133 ±4	152 ±2	149 ±3	132 ±7	135 ±6
SV	95 ±4	95 ±4	100 ±3	114 * ±4	106 b ±2	116 * ±4
CO	12.5 ±0.7	12.7 ±0.8	15.1 ±0.5	16.9 * ±0.5	14.0 ±0.5	15.6 ±0.7
MAP	111 ±4	110 ±3	114 ±4	108 ±5	110 ±5	107 ±4
RPP	220 ±10	229 ±14	278 ±8	261 ±11	217 ±15	223 ±19
SVR	728 ±49	704 ±41	606 ±14	511 ±17	646 ±49	564 ±37
SW	143 ±5	142 ±8	155 ±10	167 ±12	158 ±6	168 ±11

HR - heart rate (b·min^{-1}); SV - stroke volume (ml·beat^{-1}); CO - cardiac output (l·min^{-1}); MAP - mean arterial pressure (mmHg); SVR - systemic vascular resistance (dyn·s·cm^{-5}). RPP - rate pressure product (HR x SBP x 10^{-2}) SW - stroke work (g·m). * pre vs post data significantly different ($p<0.05$).

Table 5.8 Changes in peak torque ($N \cdot m$) following training.

	Angular Velocity ($rad \cdot s^{-1}$)							
	Extension				Flexion			
	1.05		3.14		1.05		3.14	
Knee	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Control	134 ±7	143 ±6	81 ±5	78 ±7	75 ±5	69 ±9	52 ±3	54 ±6
Cycle	147 ±11	153 ±7	77 ±5	90 * ±5	88 ±8	98 ±6	57 ±6	65 ±6
Circuit	143 ±14	156 ±15	88 ±13	115 * ±8	91 ±6	106 * ±8	63 ±6	77 * ±5
Shoulder								
Control	71 ±4	81 ±13	52 ±2	50 ±6	50 ±3	46 ±5	33 ±3	35 ±7
Cycle	66 ±7	82 * ±6	49 ±7	57 ±7	47 ±4	50 ±5	31 ±4	38 ±5
Circuit	73 ±5	90 * ±10	58 ±3	70 * ±4	50 ±2	58 ±4	37 ±3	49 * ±3

* pre vs post data significantly different ($p<0.05$).

Figure 5.1. Accumulated work over the three 20s work bouts of knee extension/flexion exercise at 3.14 rad$\cdot$s^{-1}. * = pre vs post significantly different ($p<0.05$).

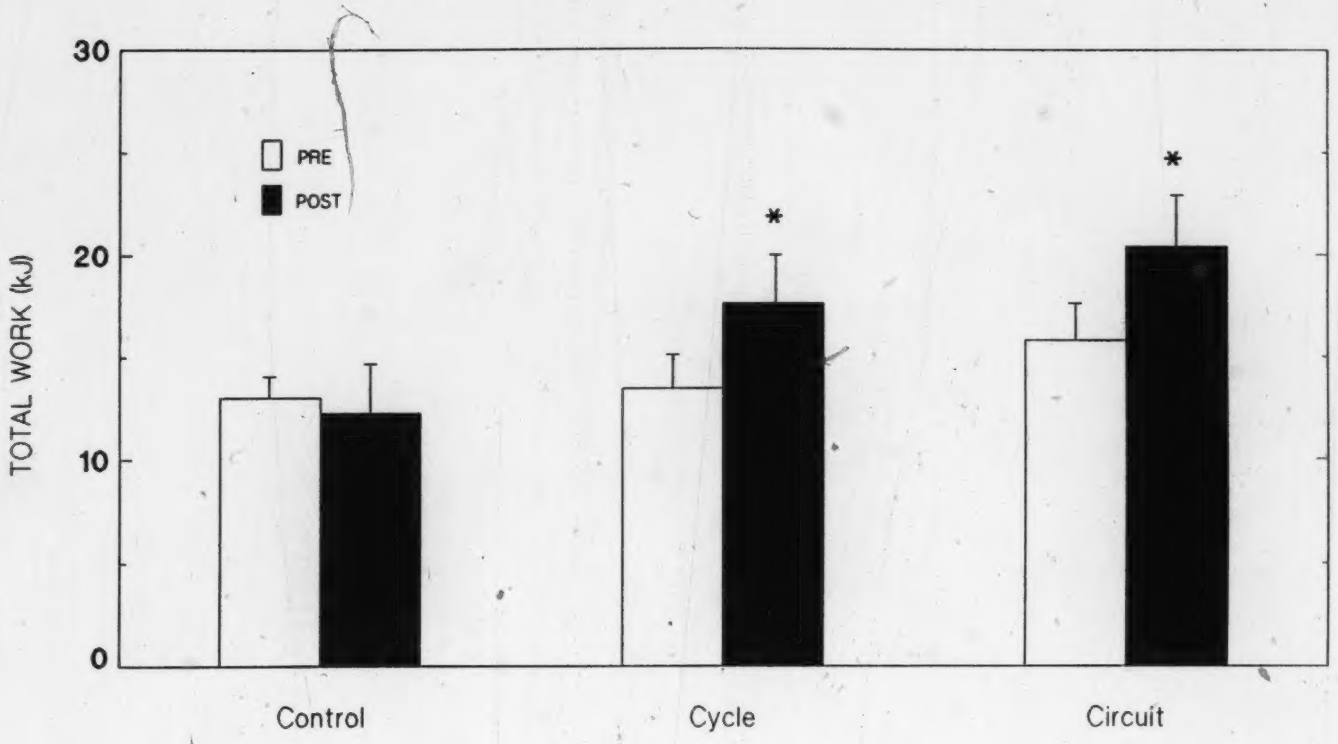

30
20
10
0
TOTAL WORK (kJ)
PRE
POST
*
*
Control
Cycle
Circuit

Figure 5.2. Accumulated work over the three 20s work bouts of shoulder extension/flexion exercise at 3.14 $\mathrm{rad \cdot s^{-1}}$. * = pre vs post significantly different ($p<0.05$).

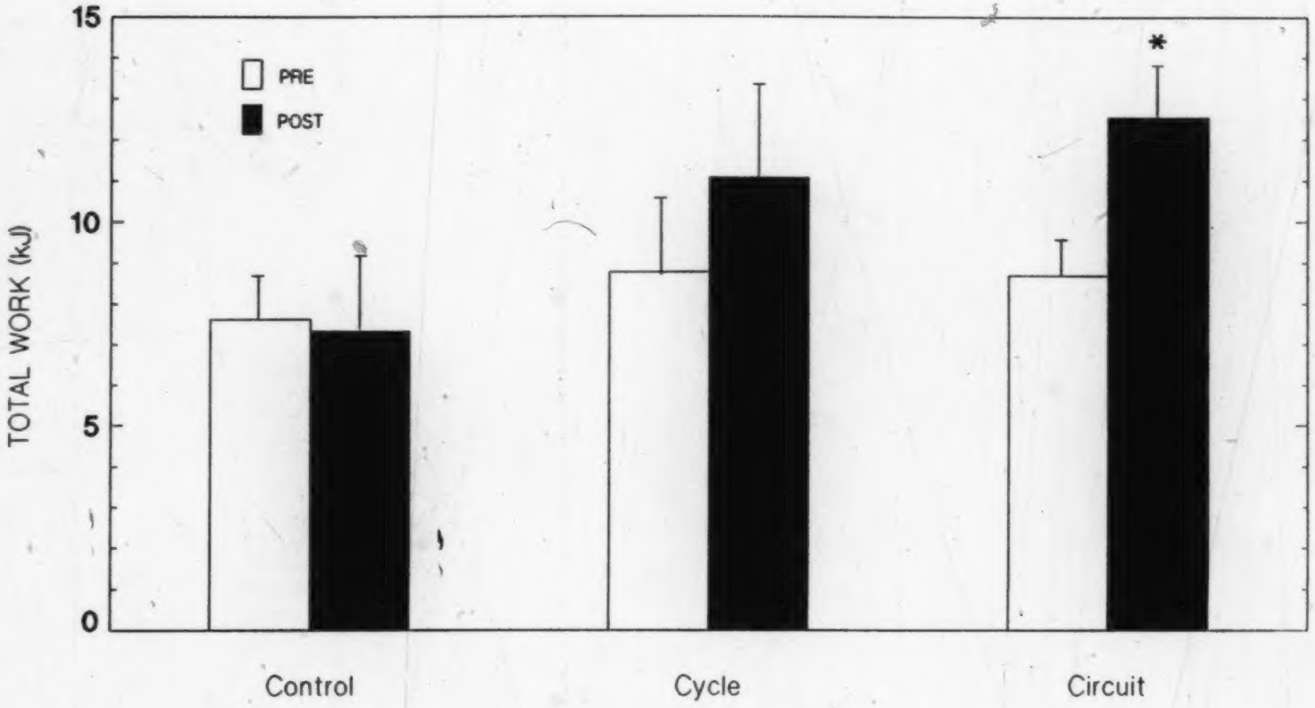
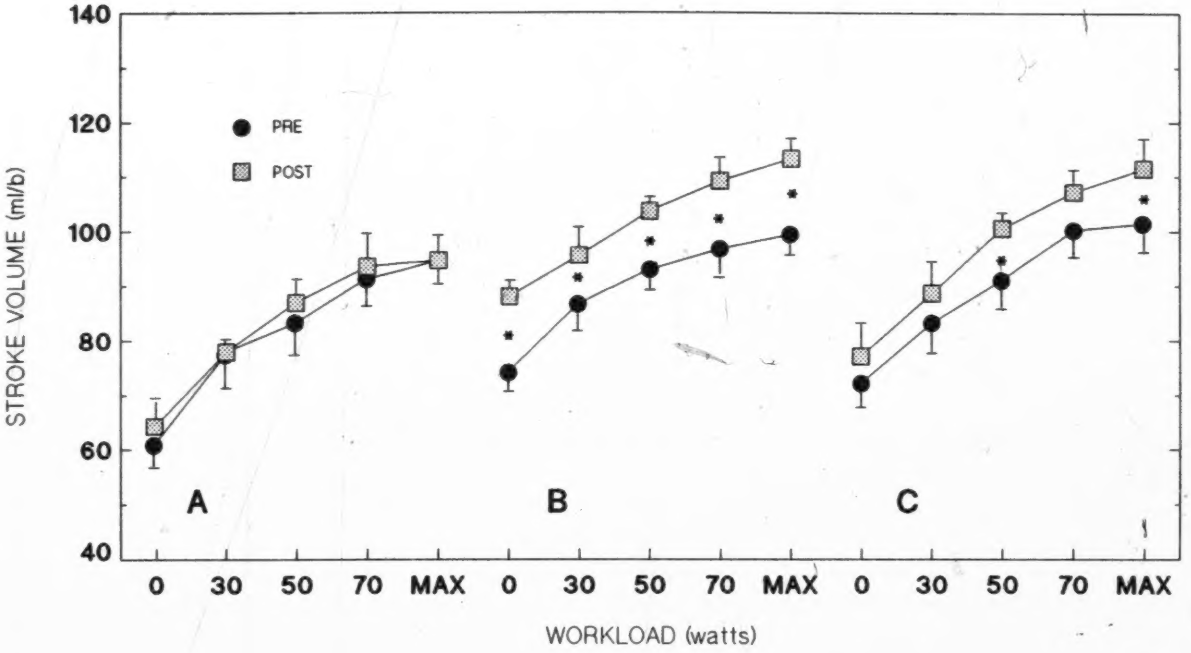

15
10
5
0
TOTAL WORK (kJ)
PRE
POST
*
Control
Cycle
Circuit

Figure 5.3. Changes in the stroke volume response to a graded exercise text on a bicycle ergometer. Panel A represents the Control group; Panel B the Cycle training group; and Panel C the Circuit training group. * pre vs post training data significantly different (p<0.05).


140
120
100
80
60
40
STROKE VOLUME (ml/b)
PRE
POST
A
B
C
*
0
30
50
70
MAX
0
30
50
70
MAX
0
30
50
70
MAX
WORKLOAD (watts)

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CHAPTER 6

GENERAL DISCUSSION

The rationale for considering hydraulic circuit training as an alternate exercise mode in cardiac rehabilitation relates to the physiological factors that limit exercise capacity in both the healthy individual, and the patient with impaired myocardial function due to occlusive coronary artery disease.

During aerobic exercise with major muscle groups in the upright position, the maximal level of exercise that can be performed is limited by two dominant factors; the ability of the cardiovascular system to deliver oxygen to the exercising skeletal muscle; and the ability of the exercising muscle to extract oxygen from the blood for use as fuel for muscular work (3,5,25). When the intensity of muscular work produces energy demands that exceed the capacity of the cardiovascular system to deliver, or the skeletal muscle to use oxygen, then exercise cannot be maintained for more than a few minutes owing to a rapid consumption of available substrate for anaerobic glycolysis, a progressive accumulation of lactate and a fall in pH, and a fall in high energy phosphate concentrations (13), all of which limit continued muscular work.

In the healthy individual, the increase in CO, that accompanies graded exercise, is attributable both to an increase in SV, and HR. The augmented SV is usually produced by an increase (compared with resting values), in both the LVEF and LVEDV (3,5). Notably, the enhancement of CO and LVEDV that occur during acute exercise is accompanied by only slight increases in the filling pressure (left ventricular end-diastolic pressure, or pulmonary capillary wedge pressure) of the left ventricle (3).

The central cardiovascular adjustments to aerobic exercise in patients with coronary artery disease typically differ from those of healthy individual in that the maximal values for CO, HR, and for MAP are lower (6,8). During submaximal exercise, the relationship between CO and VO_2 also tends to be reduced. Depending on the competence of the left ventricle, signs or symptoms of cardiac dysfunction may develop, reflected as pulmonary capillary hypertension (dyspnea), myocardial ischemia, or arrhythmia (6). Moreover, most coronary artery disease patients exhibit some signs of left ventricular dysfunction during aerobic exercise, including a decrease in SV at higher workloads (11,20,22), reduced myocardial contractility, and increased left ventricular end diastolic pressure (6). Despite the potential for developing ischemic-induced arrhythmias aerobic exercise,(walk-jog format) remains the mode of choice in most cardiac rehabilitation programs.

Left ventricular dysfunction during exercise reflects a disparity between myocardial oxygen supply and demand (5,19,28). The major determinants of myocardial oxygen uptake (MVO_2) are wall tension, the number of times each minute that tension has to be developed, and under what contractile state that amount of work is being done (27). Wall tension, in turn, is related to the pressure generation of the heart, and to the size of the left ventricle from which the pressure is being generated (Law of LaPlace). Thus, during aerobic exercise involving a large muscle mass, the increased venous return results in increased wall tension, and a greater MVO_2. Because the myocardial a-vO_2 difference is near maximal at rest, very little additional O_2 extraction can take place during exercise (5,6). Any substantial increase in myocardial oxygen demand must therefore be met by increases in coronary blood flow, which may be compromised in coronary patients.

Since the major problem in patients with ischemic heart disease is inadequate coronary blood flow, a reduction in preload should reduce MVO_2 and the risk of ischemic-induced arrhythmias. By exercising smaller muscle masses one at a time, the increases in LVEDV, ventricular radius, and consequently myocardial tension and MVO_2 would be reduced. Given the desire to limit the increase in preload, and the fact that the majority of the adaptive changes in cardiac patients occurs at the peripheral level (6,7,25), it seemed appropriate to consider a training mode which enables smaller muscle groups to work at relatively high loads without precipitating a sustained heavy demand on the central circulation.

This study was therefore undertaken to evaluate the feasibility of hydraulic circuit training as an alternate exercise mode for cardiac patients. Prior to assessing the efficacy of hydraulic circuit training in cardiac rehabilitation, an investigation of the acute cardiovascular response to strength training exercises seemed warranted. To this end, the effects of velocity and muscle mass on the acute CO and MAP response to maximal isokinetic exercise was investigated. Subsequently, the effect of a hydraulic circuit training program on the cardiovascular function of untrained middle-aged males was assessed. Finally, the acute cardiovascular response to maximal hydraulic resistance exercise was investigated in a groups of post coronary artery bypass patients, prior to their participation in a study on the efficacy of hydraulic circuit training in cardiac rehabilitation.

Acute Cardiovascular Response

The acute cardiovascular response to aerobic and strength training exercise have previously been considered to be distinctly different (17). Conventional descriptions of the hemodynamic differences implies that the mode of contraction specifically determines the response. However, the characteristic hemodynamic responses are largely based on experiments involving aerobic exercise with large muscle groups and isometric exercise with much smaller muscle groups (10,17). More recent research suggests that many features of the hemodynamic response and its regulation are common to both modes of exercise (4,16).

According to the conventional concepts the magnitude of the pressor, and HR response to strength training exercises, is proportional to the relative force and duration of the muscular contraction (17), and independent of the active muscle mass. Present findings suggest that the magnitude of the HR and MAP responses is influenced not only by the relative load but also by the active muscle mass. The increases in HR and MAP during isokinetic elbow extension/flexion exercises were qualitatively similar to those seen during maximal knee extension/flexion exercises, but the absolute values achived were smaller.

One commonly expressed concern associated with strength training exercises is that the rise in blood pressure may produce excessive myocardial stress in patients with coronary artery disease (2). The HR and MAP response to hydraulic circuit training was at a percentage of the maximum value observed during maximal cycle ergometry. While the blood pressure response to maximal hydraulic resistance exercise approached that attained during aerobic exercise, the increase may actually contribute to an increase in subendocardial perfusion and myocardial oxygen supply, offsetting some of the increased myocardial oxygen demand brought on by an increase in afterload. (14).

For many years the pressor response to high intensity strength training exercises was believed to impose excessive myocardial oxygen demands on cardiac patients. Recently this concept has been disputed, as investigators (9,12,14,26) have not found a greater incidence of arrhythmias or ischemic responses in patients with coronary artery disease performing strength training exercises, compared with aerobic exercise. Within the limits of the present study, the rate pressure products were reduced, compared to values achieved during aerobic exercise on a bicycle ergometer. Further, it has been suggested that during strength training exercises, the rate pressure product may overestimate myocardial oxygen costs (10).

The lower rate pressure products during the isokinetic and hydraulic resistance exercises reflected a lower maximal HR, secondary to a reduced muscle mass (4,16). Further, the fact that the level of cardiovascular stress was maintained for only 20s intervals during training, suggests that if ischemia is not elicited during maximal exercise on a bicycle ergometer it is unlikely to appear during hydraulic circuit training performed at submaximal repetitions with a smaller muscle mass.

Adaptation to Circuit Training.

Although it is traditional to segregate the factors limiting exercise performance into central and peripheral components, it should be recognized that these two components are intricately intertwined, and that this segregation is, to some degree an artificial one. For example, in the present study training was associated with an increase in maximal CO. This adaptation to training would generally be classified as a central adaptation, however, its morphologic basis may have been in the skeletal muscle, and not the heart. Exercise training results in increased vascular conductance and maximal blood flow which together are responsible for the increased systemic vascular conductance during exercise in the trained state. The increase in vascular conductance in trained muscles implies an attenuation in sympathetic vasoconstrictor tone of the local resistance vessels, which in turn contributes to the augmented CO. Presumably, the decreased sympathetic activation is a result of the increased oxidative capacity of the trained muscle such that the same absolute level of exercise imposes less of a relative load on the muscle's metabolic system (23,24,25).

It is intriguing that skeletal muscle capillarization both in regards to magnitude and time course very closely follows the variation observed for VO_2 max with physical conditioning and deconditioning, whereas the mitochondrial enzyme changes are much more pronounced, especially in the detraining period (1,25). The role of the capillaries is obviously to supply a surface area for exchange of gases, substrates, metabolites and heat between blood and the muscle cell. Increased muscle usage during hydraulic resistance training may stimulate a proliferation of the capillary bed, with an increase in the number and dimension of muscle capillaries and an increase in the capillary to fiber ratio (1). The primary advantage of a high capillary density in trained muscles would be to allow for an adequate mean capillary transit time at high perfusion rates (23).

Present findings suggest that hydraulic circuit training might be useful in training patients with a severe left ventricular dysfunction. Patients with left ventricular function related to coronary artery disease constitute a group of individuals particularly in need of intensive rehabilitation efforts. However, somewhat paridoxically, patients with left ventricular dysfuntion are often excluded from formal cardiac rehabilitation programs (18), which have generally focused their efforts on patients with less complicated illnesses. The rationale for excluding patients with ventricular dysfunction from cardiac rehabiliation programs has been based largely on the concern that such patients are at excessive risk for adverse events during exertion, or that exertion might adversely affect the natural history of ventricular dysfunction, precipitating or accelerating decompensation of the left ventricle.

These concerns are contrasted by present findings which suggested that hydraulic circuit training was associated with a decrease in peripheral vascular resistance. The resulting fall in ventricular afterload would be expected to allow patients with abnormal left ventricle function to increase their cardiac output and comfortably support some level of exertion.

Finally the increases in muscular strength and endurance observed with hydraulic circuit training suggests that this form of training should be considered as an alternate exercise mode for patients who require a higher degree of muscular strength and endurance for their vocational or avocational activities. By training the muscle groups involved in the specific activities a reduction in the cardiovascular stress associated with a given muscular effort may be anticipated.

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CHAPTER 7

CONCLUSIONS AND RECOMMENDATIONS

From the results of the present study the following conclusions appear justified:

1. The magnitude of the heart rate and blood pressure response to maximal isokinetic appear to be a function of the active muscle mass and independent of the exercise velocity.
2. Hydraulic circuit training was effective in enhancing the maximal aerobic power of previously untrained middle-aged men.
3. The chronic hemodynamic responses to a program of hydraulic circuit training include an increase in maximal stroke volume, and cardiac output with a relative bradycardia at absolute submaximal levels of exertion.
4. The central circulatory demands associated with maximal hydraulic resistance exercise were less than those associated with maximal exercise on a bicycle ergometer, in a group of post coronary artery bypass patients.
5. Patients recovering from coronary artery bypass surgery demonstrated an increase in maximal aerobic power, as well as muscular strength and endurance following a program of submaximal hydraulic circuit training.
6. The principal hemodynamic responses to circuit training in post coronary artery bypass patients included a relative bradycardia at rest. At a given absolute workload there was a reduction in heart

rate, a corresponding increase in stroke volume, and a decrease in mean arterial pressure.

Recommendations. Findings from the present studies implied that hydraulic circuit training was effective in enhancing maximal aerobic power of previously untrained individuals. Information on changes in skeletal muscle oxidative potential and muscle capillarization would help to delineate the mechanisims responsible for changes in systemic oxygen uptake. Further, present findings need to be investigated in other subject pools, including older, and higher fit individuals.

Regarding the clinical application of circuit resistance training as an alternate exercise mode in cardiac rehabilitation. Present findings are limited to patients recovering from coronary artery bypass surgery, these patients tend to be more stable, and their exercise training tends to progress more rapidly than post myocardial infarction patients. Thus, the use of circuit resistance training needs to be investigated further in a variety of clinical populations prior to its endorsement as a viable exercise mode in cardiac rehabilitation. Specific issues which need to be addressed include:

1. A comparison of the cardiovascular adaptations to a program of hydraulic circuit training with an interval arm ergometry program.
2. An examination of the efficacy of hydraulic circuit training in a group of patients recovering from a myocardial infarction.
3. An assessment of the acute cardiovascular response in a group of patients with impaired ventricular function to hydraulic resistance exercise.

4. An evaluation of the efficacy of hydraulic circuit training on the maximal aerobic power, muscular strength, and endurance of patients with left ventricular dysfunction.

APPENDIX A

REVIEW OF RELATED LITERATURE

In order to provide a background for further discussion this review will focus on the cardiovascular responses to aerobic and strength training exercises. For the purposes of this review aerobic exercise will be conSidered to include upright activities such as walking, cycle ergometry, and running. Strength training exercise will encompass isometric contractions, isotonic weight lifting, and isokinetic type exercise.

1. ACUTE CARDIOVASCULAR RESPONSE TO EXERCISE

With any increase in muscular activity the cardiovasular system must operate in an integrated fashion to increase muscle blood flow or the driving pressure (220). To accomplish this goal, heart rate, stroke volume, cardiac output, and peripheral resistance can be altered.

1.1 Aerobic Exercise

i) Cardiovascular Adjustments. The energy required for large muscle group exercise that lasts for more than several minutes can only be supplied by aerobic metabolism. With progressively increasing workloads there is a linear increase in oxygen uptake (VO_2) until VO_2 max is achieved (213). Thus, VO_2 is directly determined by the external power output. The VO_2 necessary to perform a particular activity, directly determines cardiac output (CO) at any level of submaximal or maximal exercise (14,206).

In a healthy individual, the increase in CO is achieved by a three fold increase in heart rate (HR) and a two fold increase in stroke volume (SV) (14,21,64,141). While linearly related to VO_2 and CO, the increase in HR is not directly determined by the external load. Rather, HR is correlated to the relative load, expressed as a percentage of maximal voluntary contraction (MVC) or percent VO_2 (35,216). The initial increase in HR during aerobic exercise is due to vagal withdrawal as a result of changes in the activity of the higher autonomic centers (221). Since the HR increases with the onset of exercise, it is probable that signals from both voluntary higher motor centers of the cerebral cortex, and muscle afferent impulses influence the excitatory state of the autonomic centers, rather than a reflex arising from muscle metabolites or from humeral agents (10,138,157). The subsequent slower increase in the HR as exercise progresses results from an increase in sympathetic drive. This increases is due to afferent impulses arising from the metabolic receptors in the active muscles and an increased level of central command, as local muscle fatigue progresses (157,161).

During aerobic exercise, the SV reaches a maximum value at approximately 40-50% VO_2 max (or a HR of approximately 110-120 $beats \cdot min^{-1}$) (14). In healthy individuals once the maximal SV is achieved, it is preserved for the duration of the exercise (21). The normal SV response represents the utilization of both a diastolic and systolic reserve mechanisms (141,239). The presence of the Frank-Starling mechanism has been demonstrated during upright cycle ergometry. Hemodynamic studies have shown an increase in left ventricular filling pressure with increasing exercise (46,187). Echocardiographic and radionuclide studies have documented increases in left ventricular end-diastolic volume (LVEDV), and decreases in left ventricular end-systolic volume (LVESV), with a resultant increase in SV and left ventricular ejection fraction (LVEF) during exercise (47,187,226).

Enhanced contractility is exhibited, during exercise in the intact heart, by a decrease in LVESV (187), an increase in the LVEF of 5% or more, and an increase in ejection rate (239). A decrease in afterload or systemic vascular resistance also enhances systolic emptying (239). Thus, during aerobic exercise involving a large muscle mass, both the Frank-Starling mechanism, as well as an enhanced contractile state provide for the exercise related increase in SV and LVEF (164,244).

During large muscle aerobic exercise, systolic blood pressure (SBP) increases linearly with increments in workload, there is a slight decrease or no change in diastolic pressure (DBP) and only a moderate increase in MAP (22). The change in MAP result from changes in CO and systemic vascular resistance. The increase in MAP seen during exercise is not directly determined by the external work rate, but is correlated to the relative workload (35). While CO is related to the external load, regardless of the size of the active muscle mass, exercise induced vasodilation is dependent upon muscle mass. As a result, an increase in CO produces a higher blood pressure during exercise involving a smaller muscle mass (15).

The distribution of CO during aerobic exercise has received conciderable attention. It is now recognized that the augmented blood flow to exercising muscle is accomplished not only by an increase in CO, but also by redistribution of blood flow. With moderate to severe aerobic exercise, blood flow to the active muscles increases and is accompanied by an elevation of coronary blood flow (175,219). Splanchnic and renal flow remain at pre-exercise levels despite increased vasoconstriction in these visceral beds (36). Blood flow to the non-exercising muscles is diminished (175). The cerebral circulation is maintained during moderate aerobic activity, but there is a tendency for this flow to diminish slightly during maximal aerobic exercise as a result of the fall in arterial carbon dioxide tension that accompanies hyperventilation (35). Thus, vasodilation occurs in the arteriolar beds of the exercising muscle and the heart, while vasoconstriction takes place in the gut kidney and, to a lesser extent, in the skin and resting muscle.

The normal central and peripheral circulatory adjustments to aerobic exercise in an healthy individual can be recapitulated as follows: CO increases in direct proportion to the augmentation of VO_2. The increased CO is mediated by increases in both HR and SV, and is directed to exercising skeletal muscles, the myocardium, and with continued activity to the skin. Blood flow to non-exercising muscles is reduced due to a general sympathetic vasoconstriction. At submaximal levels of exertion, muscle blood flow per unit tissue, the degree of peripheral vasoconstriction, and the increase in HR are all functions of the relative VO_2.

ii) Cardiac Patients. There are several contrasting features to the cardiovascular response to aerobic exercise in patients with coronary artery disease. The response to aerobic exercise is dependent upon the extent of the disease, the functional capacity of the remaining myocardium and the neurohormonal status (26). While both HR and CO usually increase with increments in external load, the maximal levels achieved are often attenuated, compared to the values achieved by age-matched healthy individuals (36). The SV response is quite variable and has been reported to increase, or remain unchanged with the transition from rest to peak exercise (95,195,196).

The degree of left ventricular dysfunction during aerobic exercise is quite variable. Dehmer et al (55) reported an increase in LVEDV, a decrease in LVESV, and an increase in LVEF during exercise in patients with angiographic normal coronary arteries, or one vessel disease. In patients with two and three vessel disease, however, there was a decrement in ventricular function. The LVESV instead of deminishing actually increased, suggesting that the left ventricle was unable to reduce the end-systolic volume against the elevated afterload of exercise. Since the LVESV increased as much as the LVEDV, the SV remained fixed at the pre-exercise level, and LVEF fell. Similar results were reported by Carroll and associates (31) who noted a decrease in LVEF among patients who experienced ischemia during exercise. Compared to the pre exercise values, these patients exhibited an increase in LVEDV and a disproportionate increase in LVESV resulting in either a decrease or no change in the SV at peak exercise. Ehsani and coworkers (61) reported on a group of post infarction patients who were able to attain a true VO_2 max. Again results were variable. In patients who had an abnormal LVEF, the LVESV was increased. A normal LVEF response was associated with a reduction in LVESV at peak exercise.

Peripheral circulatory regulation in patients with coronary artery disease is, in principle, similar to that seem in healthy individuals (35). However, it has been noted that during submaximal aerobic exercise muscle blood flow is attenuated, and that maximal muscle blood flow is lower (36). A reduction in maximal muscle blood flow is consistent with a higher level of systemic vascular resistance (36,42), and may attributed to increased sympathetic tone (70).

In summary, the circulatory adjustments to aerobic exercise in patients with coronary artery disease differs from that of the healthy individual in that the maximal values for VO_2, CO, HR, and MAP are lower. During submaximal exercise, the relation between VO_2 and CO tends to be reduced (36). Moreover, some patients exhibit signs of left ventricular dysfunction during exercise, including a decrease in SV with increments in workload, reduced myocardial contractility, and increased left ventricular end-diastolic pressure (36). The peripheral circulatory regulation essentially corresponds to that seen in healthy individuals.

1.2 Upper Body Exercise

i) Cardiovascular Adjustments. While both upper and lower body exercise share the same control mechanisms, many of the the cardiovascular responses (at a given power output, or VO_2) differ

(13,20,236,237).

The CO, at a given VO_2, is similar for arm and leg exercise (13,20,50,153,192). Miles and coworkers (155) used a variety of techniques (dye-dilution, direct Fick, CO_2 rebreathing, electrical impedance) to investigate the relationship between VO_2 and CO during submaximal arm cranking exercise. Their analyses indicate that CO increases from 5 to 7 $l \cdot min^{-1}$, per liter of VO_2 during submaximal arm exercise. During maximal exercise, however, the CO values were lower for arm exercise than for leg exercise (155). Reybrouck and coworkers (192) found that maximal CO was approximately 30% lower during arm cranking than cycle exercise. Not suprisingly, this disparity in maximal CO corresponds to the difference in peak VO_2 between arm and leg exercises (207,236).

Although the CO is similar for arm and leg exercise at a given absolute (submaximal) workload, it is achieved by marked differences in HR and SV. Investigators consistently report higher HRs and lower SVs during arm cranking than during cycle exercise (13,20,39,50). The elevated HR reflects a greater sympathetic stimulation during arm exercise (49). An increased sympathetic stimulation should improve myocardial contractility (224); however, similar values of contractile indices have been reported for arm and leg exercise (153). An increase in myocardial contractility may not be detected during arm exercise because of differences in cardiac filling and preload. During upper body exercise, the reduced skeletal muscle pump yields a smaller venous return and consequently LVEDV (36). Therefore, a reduced preload will have the myocardium contracting on a less efficient portion of the ventricular function curve. Under such conditions, increased sympathetic stimulation would be needed to obtain similar contractility values during upper body as compared to lower body exercise (153). This reduced preload hypothesis may partially explain why SV does not increase markedly during arm exercises (20,145,154).

During arm and leg work, most of the increase in CO is directed towards the active skeletal musculature. Vasodilation occurs in the active musculature because of the local accummulation of metabolities such as K^+, H^+, as well as osmolality changes, and hypoxia (36,156). This local control supersedes the constrictor effects of the sympathetic output associated with exercise (219). Therefore, the greater the skeletal muscle metabolic rate, the more metabolites produced and the greater the blood flow. According to Clausen's (36) calculations absolute muscle blood flow is similar during aerobic arm and leg exercise, performed at the same VO_2. However, since upper body exercise involves a smaller skeletal muscle mass, blood flow is higher for a given muscle mass (37).

The hemodynamics of upper and lower extremety exercise are quite different. According to Poiseiulle's law, flow is the quotient of driving pressure divided by resistance. Numerous investigators report higher systolic (13,153,237) diastolic (13,153) and aortic (20) pressures during arm exercise. In order to elicit the same CO, resistance must therefore be proportionally higher during upper body exercise. It seems reasonable that upper body exercise might generate sufficient intramuscular tension to mechanically increase peripheral resistance within the exercising arms. Exercise performed with a smaller skeletal muscle mass needs to develop a greater percentage of its maximal tension to produce a given power output (207). This might result in intramuscular tension that exceeds perfusion pressure and thereby effectively decreases the vascular cross-sectional area perfused. A smaller vascular cross-sectional area being perfused by the same CO will result in a greater resistance. Further, Davies et al (49), reported that plasma catacholamine concentrations are inversely related to skeletal muscle masss during submaximal exercise at a given VO_2. Therefore, vasoconstrictor drive may be expected to be greater during arm exercise than leg exercise.

An isometric exercise component (114) has been proposed as mechanisms leading to a greater vasoconstrictor tone and elevated total peripheral resistance during arm work. In comparison to cycle ergometry, arm cranking exercise has a greater isometric component for torso stabilization, and grasping the handcrank (37,39,207). The imposition of an isometric exercise component upon a aerobic exercise elevates the blood pressure response above levels elicited by aerobic exercise alone (114,125). This pressor response is believed to be a reflex from the contracting skeletal muscle (120,149), which results in increased vasoconstrictor tone (162). Therefore, the greater the isometric component in upper body exercise the greater the total peripheral resisitance and blood pressure.

ii) Cardiac Patients. Patients with coronary artery disease perform conciderably less external work before experiencing angina when aerobic exercise is performed with the arms rather than with the legs (40,132). This phenomenon may be attributed to the higher HR and SBP responses during arm exercise compared with leg exercise at a given submaximal workload. Since HR x SBP is an index of myocardial oxygen demand (126), myocardial oxygen uptake (MVO_2) at a given workload has been assumed to be higher during upper body exercise. This physiologic response is believed to be due to many factors, including increased sympathetic outflow during arm exercise, compensatory tachycardia secondary to a reduced SV, concomitent isometric contraction, and vasoconstriction in the inactive skeletal muscles (13,237).

1.3 Strength Training Exercises

i) Cardiovascular Adjustments. Isometric exercise has been recognized as an important component of strength training, receiving its greatest impetus as a conditioning method when it was demonstrated that 6s contractions at two-thirds of MVC promoted significant increases in strength (96,138). Later however, it was shown that these strength gains were limited to the conditioned angle of the joint (138). Cardiovascularly, isometric exercise poses a different stress than aerobic exercise. Aerobic exercise increases CO significantly with only a moderate effect on MAP. With an isometric contraction, the CO, contractility of the left ventricle, HR, and systemic arterial pressure both systolic and diastolic are increased (138,220).

During an isometric contraction, intramuscular pressure is increased, compromising muscle blood flow even at low tensions (9).

Because of the failure of local muscle blood flow, exercising muscles must work under partial or complete ischemic conditions. Therefore, only a modest increase in VO_2 is seen. As with aerobic exercise there is a tight coupling of VO_2 and CO, consequently there is only a modest increase in CO during isometric exercise (9,25,138,220). The increase in the CO is mediated primarily by an increase in HR. However, even with conciderable muscle tension, the peak value for HR is still modest compared to aerobic exercise (139,186). The initial increase in HR at the onset of the static contraction is due to vagal withdrawal (138,220). As the isometric contraction progresses further increases in the HR are likely the result of symathetic stimulation secondary to increased activation of the metabolic receptors in the ischemic muscles, and "central command" (as muscle fatigue demands an increasing voluntary effort to maintain a constant force) (138,220).

During isometric exercise the SV tends to be maintained at, or near pre-exercise levels. The normal left ventricle is able to maintain the SV at resting levels with little or no increase in end-diastolic pressure, despite the marked increase in SBP and afterload (220). These findings are consistent with enhanced myocardial contractility (9,156,220). Both LVEDV and LVESV increase slightly; however decreases in one or both have also been noted (220). Any increase in the SV during an isometric contraction is likely due to increased venous return secondary to venoconstriction and the squeezing out of venous blood from the muscles at the onset of the contraction (9). The LVEF tends to remain the same during isometric exercise (142). Thus, the maintenance or slight increase in the SV that occurs during isometric exercise is a result of an increase in the relative preload and an increase in contractility.

The blood pressure response to isometric exertion has recieved conciderable attention (9,25,27,138,143). Systolic, diastolic and mean arterial blood pressure increase in a roughly parellel manner at the onset of isometric exercise (139,140). The blood pressure usually reaches a steady state within a few minutes at low levels of exercise, whereas there is a continual rise during sustained contractions greater than 15-20% of maximum voluntary contraction (MVC) (139,140). Mean arterial pressure is proportional to the product CO and systemic vascular resistance. During isometric exercise systemic vascular resistance is unchanged or increases slightly (9,139,156), consequently the increase in MAP is primarily a result of the increase in CO (27).

According to clasical concepts the magnitude of the pressor and the HR response is closely linked to relative effort, or developed force, expressed as a fraction of MVC (139,148). However, recent studies have demonstrated that the magnitude of the cardiovascular response is greatly influenced not only by relative load but also by the active muscle mass, and the absolute force generated (25,137,158). This relationship is nonlinear, with decreasing effects of increasing active muscle mass at high levels of force development (23).

ii) Cardiac Patients. Although coronary patients are generally cautioned to avoid isometric exercises, it appears that they may be less hazardous than was once presumed. Dispite earlier reports (16,28) that isometric exercise may lead to arrhythmias, ventricular decompensation, or sudden death, more recent studies (52,53,66) indicate that nonsustained isometric exercise, regardless of the percentage MVC used, generally fails to produce angina, ST-segment depression, or significant ventricular arrhythmias among select patients.

Studies on combined static/dynamic exercise have shown that the ischemia threshold is higher as compared to aerobic exercise alone (52,88,123,217). Sheldahl et al (218) evaluated the cardiovascular responses to carrying graded weight loads in a group of post-myocardial infarction (MI) patients. The peak DBP was 110 mmHg, whereas the peak HR, VO_2, rate pressure product, and frequency of ischemic responses were significantly lower during load carrying than those observed during the aerobic exercise alone. Increased subendocardial perfusion, secondary to the elevated DBP, may contribute to the lower incidence of ischemic responses reported during the combined static/dynamic effort (66,123).

One of the major concerns regarding isotonic weight training in cardiac patients is the expected increase in blood pressure. Extreme increases in both SBP and DBP have been reported during near maximal weight lifting in body builders (144). While studies by Fleck and Dean (65) and Freedman et al (71) have also reported significant increases in blood pressure with high intensity weight lifting exercises, the values were lower than those reported by MacDougall et al (144).

Vander and colleagues (234) recently reported on the safety and feasibility of using circuit weight training (CWT) as part of a cardiac rehabilitation program. They noted that the DBP reached a peak of 103 mmHg during CWT, at 40% of one repetition maximum (1RM). Although this level of DBP is frequently used as an end point to aerobic exercise, it hasn't been established whether an increase in DBP to this magnitude constitutes excessive myocardial risk for patients with coronary artery disease. As pointed out by Vadner et al (234), CWT elicited small increases in HR, rate pressure product, and MAP. The peak HR and rate pressure product was only 64 and 62% (respectivelty) of the maximum values observed during a symptom limited graded exercise test. No significant arrhythmias, abnormal hemodynamics, ST-segment depression, or symptoms were recorded during the exercises. Similarly, Saldivar and coworkers (200) reported that circuit weight training program was not associated with symptomatology, ST-segment changes, or arrhythmias in patients with coronary artery disease.

These findings suggest that the myocardial oxygen supply/demand relationship may be altered during weight lifting exercises so that the incidence of significant arrhythmias, ST-segment depression, or symptoms are reduced (52,123). If ischemia is not elicited by maximal aerobic exercise, it is unlikely to appear during strength training exercises performed at submaximal intensities. These observations are changing the cautious attitude toward strength training, particularly in regard to vocational counseling and exercise prescription for cardiac patients (66).

2. CARDIOVASCULAR CONTROL

Regulation of the cardiovascular system involves many mechanisms, some of which are important in short term regulation, whereas others are important in long-term regulation. The rapid changes in HR and blood pressure at the onset and cessation of exercise would seem to implicate neurogenic mechanisms as the regulators of the cardiovascular system during exercise (157). While the neural mechanisms responsible for the changes in autonomic efferent activity to the heart and the blood vessels are not completely understood, two general theories of neural control have evolved. In one theory the cardiovascular response to exercise is thought to be due to neural impulses, arising from the central activity that recruits motor units. The second theory is that muscle contraction stimulates afferent endings within skeletal muscle which inturn reflexly evokes the cardiovascular changes (3).

In general, the neural regulation of the cardiovascular system operates on the principle of feedback, meaning that some variable will be sensed and this information will be integrated into an effective output from the nervous system to correct the changed variable (227). Under normal conditions muscular activity is initiated by centers in the cerebral cortex that are believed to have direct input into the areas of the brain stem controlling the cardiovascular system (85,104,151,214). Thus, changes in the cardiovascular system will be linked to changes in the motor output from the cerebral cortex. This concept has been termed central command (156).

2.1 Central Command.

When partial curarization was administered to human subjects, to reduce the strength of the execising muscle, the HR and MAP response to aerobic exercise was greater than during aerobic exercise without curarization, even though the workload was the same for both contractions (11). This finding led the authors to suggests that the cardiovascular response appeared to be related to the greater motor command needed to achieve a given power output.

The effects of partial neuromuscular blockade on the HR and MAP response to static exercise in man was studied by Leonard et al (133). In this study, HR and MAP were measured while subjects performed static contractions with the knee extensor muscles. The MVC of the knee extensors was obtained and subsequent static contractions were performed either at the same absolute or relative force. The subjects were studied before and after neuromuscular blockade with either decamethonium or tubocurarine (preferentially blocks fast twitch and slow twitch fibers respectively), titrated to reduce MVC to 50% of the control value.

The MAP was greater during partial neuromuscular blockade with decamethonium than during a control contration. After the contraction was over MAP decreased immediately. Similar results were obtained after neuromuscular blockade with tubocurarine. The finding that HR and MAP were greater during the contraction at the same absolute force during neuromuscular blockade suggests that central command played an important role in determining the cardiovascular response (133). This conclusion was further supported by the fact that the static contraction at the same relative force after neuromuscular blockade elicited the same cardiovascular response when the force was only half of that observed during the control static exercise.

From the viewpoint of central command, neural regulation of the cardiovascular system is activated or altered by central neural structures to supply blood to the active tissue. Feedback from sensors located in the cardiovascular system or other areas is not integrated into this concept (227). However, evidence has accumulated to suggest that central command should incorporate signals returning from either the active muscle or the circulatory system to modulate the overall cardiovascular control.

2.2 Exercise Pressor Reflex

The exercise pressor reflex, broadly defined, comprises all the cardiovascular changes reflexly induced from the contracting skeletal muscle that are responsible for the increase in blood pressure (157). The hypothesis that cardiovascular changes during exercise are due to a reflex originating in the contracting muscle was proposed by Alam and Smirk (3), who showed that, with the circulation to the limb arrested by inflation of a pneumatic cuff, there was an increase in MAP which was proportional to the severity of the exercise. The MAP rose prior to any sensation of pain, remained elevated when the exercise ceased and returned quickly to control values only after deflation of the cuff. Subsequent studies (1,11) have substantiated the contention that a primary cause of the rise in MAP during exercise is a reflex originating in the active muscle.

The classification of afferent fibers with endings in skeletal muscle has been based both on anatomical and electrophysiological measurements. Four groups of muscle afferents have been identified, and are classified by their conduction velocity, and diameter. Of the four groups, stimulation of the group I and II larger myelinated fibers, which carry impulses from receptors located in the muscle spindles, Golgi tendon organs, joints and ligaments, and sense motion, have little effect on the cardiovascular system (160). Conversely, activation of the group III small myelinated (conduction velocity of 2.5-3.0 $m \cdot s^{-1}$), and the group IV unmyelinated fibers (conduction velocity <2.5 $m \cdot s^{-1}$) have been implicated in the reflex increase in HR and MAP during muscular contraction (120,148,149,160). Some of the group III and group IV fibers can be activiated by ventral root stimulation causing static muscle contraction (120), and by gently stroking the receptor fields of these fibers (121). Both group III and IV fibers have also been shown to respond to rhythmic contractions of muscle (119), suggesting a potential to transmit information to the spinal cord under all types of muscular exercise.

Attempts have been made to categorize some of the group III and IV muscle afferents into two groups, ergoreceptors and nociceptors. Ergoreceptors are those group III and IV afferents that cause the exercise pressor reflex. Nociceptors are those group III and IV afferents that transduce the sensation of muscle pain and therefore should be stimulated by algesic chemicals and by vigorous pinching of the muscle (128). These two catagories are likely to represent two poles on a continuum, with most of the group III and IV afferents lying somewhere in between.

Results from a study by Kaufman et al (120) suggested that although both group III and IV muscle afferents contribute to the reflex cardiovascular increases evoked by static exercise, activation of the majority of the group III fibers was associated with the mechanical efforts of the muscular contraction. Three lines of evidence supported this suggestion. First, the group III afferents were stimulated by stretching the muscle. Second, their firing increased as the tension developed by the contracting muscle increased. Third, they were stimulated by nonnoxious probing of their receptive field. The rapid response to contraction by the majority of the group III afferents has led to the suggestion that these fibers along with the central neural mechanism, are responsible for the initial increase in HR at the onset of exercise (120).

Many of the group IV afferents appear to be capable of functioning both as ergoreceptors and nociceptors. The discharge pattern of the group IV afferents stimulated by muscular contraction may indicate whether these afferents respond to mechanical or metabolic events in the muscle. One type of group IV afferents respond to contraction within 2s of the onset of a muscular contraction (119,120,128). These group IV ergoreceptors are likely to responed to mechanical events occuring in the contracting skeletal muscle. A subgroup of the group IV afferents respond to muscular contraction only after a delay of between 5 to 15s, after the onset of the muscular contraction (121,128). The discharge pattern of these group IV afferent suggest that they are likely stimulated by metabolic factors occuring in the contracting muscle (43). This suggestion is consistant with the fact that group IV fibers have "free" nerve endings within the connective tissue in muscle and hence, are well situated to detect metabolic changes in the tissue spaces (219). Further some of the group IV fibers increase their firing rate as the contraction period progresses, a finding that is consistant with the notion that they were stimulated by the buildup of metabolites in the contracting muscle (121). Consequently the group IV fibers have been proposed as the primary feedback system from the contracting muscles (156).

The metabolic stimuli for this feedback system may include K^+ ion flux from the active muscle (129,151,198). There is some evidence suggesting that K^+ release during exercise activates the group IV muscle afferents with reflex induced increases in HR, CO, MAP, and myocardial contractility (149). In man, the time course of the cardiovascular response parellels potassium release (202), similarly interstitial tissue concentrations of potassium, during exercise, and after intra-arterial potassium infusion produce similar cardiovascular responses (156,198).

Kaufman et al (119) suggested that the cardiovascular response to a muscular contraction is amplified when the arterial supply is occluded. In this study, the investigators demonstrated that the firing frequency of a population of group III and IV muscle afferents was higher during static contractions under ischemic conditions than by normal contractions at the same tension. This finding suggests that during light non-ischemic exercise the metabolic reflex neural mechanism may not be activated, however, during high intensity exercise that restricts blood flow, the metabolic reflex neural mechanism may signal a flow error that is important in eliciting the level of efferent autonomic activity to the heart and blood vessels.

Since the cardiovascular reponse occurs in the absence of a central (11) or a peripheral mechanism (111), it is apparent that both mechanisms can elicit the cardiovascular response. These mechanisms are not mutually exclusive, and to argue their relative importance in determining the cardiovascular response to exercise seems of little value. These two systems are probably redundant and integrate with other central mechanisms in the regulation of the response to exercise. The initial response to exercise is probably determined by the central recruitment of motor units and peripheral activiation of ergoreceptors connected to skeletal muscle afferents (group III). Peripheral mechanisms also provide feedback, via receptors connected to group IV muscle afferents, as to the efficacy of blood flow to meet metabolic demands (181,197). Although probably inactive during light exercise, metabolic peripheral neural mechanisms may sense flow errors and trigger efferent autonomic activity to the heart and blood vessels during moderate and high intensity exercise. As a result of both central and peripheral inputs to the cardiovascular centers, parasympathetic activity to the heart decreases and the sympathetic activity to the heart, blood vessels, and adrenal medulla increases (197,220).

2.3 Role of Muscle Mass

Since the responses of the cardiovascular system during muscular exercise are due to both central and reflex stimuli, studies have been devised to examine whether the reflex from the active muscles is related to the proportion of maximal tension achieved, or to the mass of the contracting muscle.

Lind et al (140), and Lind and McNicol (139) provided evidence to warrant the conclusion that the important factor controlling the increases in HR and MAP, associated with muscular contractions in humans, was the proportional tension exerted by the muscles. Neither the muscle mass involved, nor the absolute tension was important to these cardiovascular responses. More recent evidence takes issue with this claim (159). Mitchell et al (158) noted that the pressor response to sustained contractions at 40% MVC was progressively greater with increments in the mass of muscle. Also, the level of blood pressure following the contraction, when the circulation was arrested, was directly related to the muscle mass. This suggests that the magnitude of the active muscle mass is an important determinant of the cardiovascular response to an isometric contraction. Blomqvist et al (25) systematically examined the relationship between active muscle mass and the cardiovascular response during aerobic exercise. Their data showed a gradual transition from an aerobic to a static hemodynamic pattern as the active muscle mass decreased. A subsequent study (136) confirmed that the HR and blood pressure responses are virtually identical during static and dynamic exercise performed with small muscle groups to a commom end point of local muscle fatigue.

Petrosky and coworkers (184,185) suggested that the exercise pressor reflex is dependent upon activation of fast twitch muscle fibers. They showed, in experiments on anesthetized cats, that during static contraction of the soleus muscle, there was no pressor response, whereas stimulation of the fast twitch motor units of the medial gastrocnemius muscle was associated with a potent pressor reflex. These data imply that the different muscle fibers have different numbers and/or types of ergoreceptors but, the possibility that the distribution of these receptors is muscle specific rather than linked to fiber type should not be rule out.

Although there are obvious quantitative differences in the cardiovascular response to aerobic and strength training exercises, the same local, peripheral, amd central reflex, humeral, and central nervous factors appear to opperate in both modes of exercise. As noted by Shephard et al (220), various combinations of central command and reflexes from the active muscles may be the primarily cause of adjustments in the autonomic outflow during both aerobic and strength training exercises. The overall hemodynamic response may differ depending on the modulating influences of various other factors, including: the number of muscles involved in the exercise and the degree of dilation of the resistance vessels in the vascular beds; the degree of constriction in the vasculature of nonexercising tissues; the dilation of the cutaneuos vessels to meet the demands of thermoregulation (169); as well as variations in the levels of circulating catacholamines (33), and arterial and cardiopulmonary mechanoreceptors (59).

3. Cardiovascular Adaptations To Training

3.1 Healthy Individuals

i) Oxygen Uptake. Adaptation to increased levels of physical activity represents a basic phenomena in exercise physiology. The single most characteristic feature of this adaptation is an increase in VO_2 max. This increase occurs in vertually all populations, from cardiac patients (36) to elite atheletes (64). Suffice it to say that response is predictable (37,102,205), related to the training load, particularly with reference to the rate of increase in VO_2 max (15), and is inversely proportional to the pretraining VO_2 max (18,90,206,213).

Viewed in terms of the Fick equation, any increase in VO_2 max can be accounted for by an increase in the arteriovenous oxygen difference (a-vO_2 diff) and CO. The increase in systemic a-vO_2 diff is the result of an improved distribution of the CO and more efficient utilization of oxygen by the active tissues (38). Capillary volume is increased in trained muscle facilitating oxygen extraction as a result of increased capillary density and decreased diffusion distance (8,24). Although the increase in systemic a-vO_2 diff may account for up to half of the improvement in VO_2 max produced by short term training programs, the increase usually accounts for only a small part of the large difference in VO_2 max between athletes and sedentary subjects and is quantitatively less important than the increase in CO (24,35,213).

ii) Cardiac Output. The ultimate VO_2 max attained by an individual, as well as the extent of increase in VO_2 max with training appears to be related to, and at least partially determined by, the maximal CO. The increase in maximal CO in response to training in healthy individuals is well documented (64,91,205,206). This increase is achieved by a markedly increased SV and a slight decrease in maximal HR following training (24). In healthy individuals, the increased SV may be attributed to either an increase in cardiac dimensions, or improved functional performance of the left ventricle (24).

iii) Cardiac Dimensions. Cardiac enlargement is a well recognized adaptation to physical training, and is associated with an increase in cardiac mass as a result of increases in wall thickness, chamber size, or both, depending on the nature of the training program (24,35).

Numerous animal studies have shown that endurance trained animals have a higher heart-weight to body-ratio than inactive members of the same species (86,213). An increase in the heart-weight to body-weight ratio, however, cannot be taken as evidence of myocardial hypertrophy. Exercise training often causes a decrease in body weight that may be responsible for the increase in the ratio (213). However, several studies have provided evidence of true cardiac hypertrophy, with an increase in absolute cardiac weight and enlargement of myocardial cells. Though variable results have been reported, aerobic training in the rat (98,172,178,210), and dog, (30), and isometric training in rat (113), and cat (168) have been shown to resulted in an increase in cardiac mass and myocardial hypertrophy.

The myocardial hypertrophy that occurs as a result of dynamic or isometric exercise training involves an increase in myocardial fiber diameter (30,168). The mode of exercise (running vs swimming), and the age and sex of the experimental animals appears to be significant determinants of the degree of the cardaic hypertrophy (168,178,210). Further, there appears to be a correlation between the intensity and duration of training and the extent of hypertrophy (168,213). Interestingly, several studies have shown that repeated development and regression of the exercise-induced cardiac hypertrophy is possible with successive phases of training and detraining (98,101). Although cardiac enlargement is a well recognized adaptation to physical training, myocardial hypertrophy, or an increase in muscle mass is not an obligatory component of the training effect (172,213).

Several studies have used echocardiography to evaluate dimensional changes on the left ventricle consequent to training in man. The echocardiographic evidence of cardiac alteration most commonly consists of an increase in left ventricular end-diastolic diameter (LVEDD) consistant with volume overload hypertrophy. Significant differences in both wall thickness and chamber size have been demonstrated between sedentary subjects and atheletes (4,233). Morganroth et al (167) and Keul et al (124) reported that endurance trained athletes had a significantly increased LVEDD without significant changes in wall thickness, wherease strength trained atheletes demonstrated significantly increased left ventricular wall thickness without major changes in LVEDD. The magnitude of difference between atheletes and sedentary subjects in left ventricular mass may however, be attributable to selection rather than to training per se (249).

Short-term (<20 weeks) endurance training programs have reported either a small increase in LVEDD and left ventricular mass (2,54,63,97,99,243), or no change in these measures (146,183,193,232, 250), dispite similar increases in VO_2 max. Of the studies that reported minimal cardiac changes with training, Wolfe et al (250) reported a significant increase in the derived SV, whereas Ricci et al (193) noted a small but significant increase in calculated left ventricular mass. Similarly, the calculated left ventricular mass was reported to be increased significantly with arm or leg aerobic training (232). In the study by Ricci et al (193) the echocardiographic results of subjects who underwent strength training were similar to those reported for runners in the same study; that is, a slight but significant increase in left ventricular mass without significant changes in wall thickness or dimensions.

In all three of the forgoing investigations (193,232,250), minimal but significant changes occurred in derived indices calculated from measured data that were not significantly altered. The interpretation of the investigators in each instance were that the changes observed probably related to the training bradycardia and small secondary increase in LVEDD rather than to an intrinsic modification of cardiac structure. It was further asserted that the training state could be attained without echocardiographic evidence of cardiac adaptation.

The variability in findings between individual echocardiographic studies may be related to several factors, including exercise mode; intensity and duration of training (142); the age of the subjects, inasmuch as responsiveness to training may be inversely related to age (249); anatomical characteristics of the chest, and differing echocardiographic techniques employed (249). While a clear relationship between short term exercise training and the degree of change in ventricular dimensions has not been confirmed, the significant decrease in left ventricular mass and dimensions that has been shown to occur during detraining studies (63,97,99) argues in favor of a hypertrophic response of the myocardium to short term aerobic training. This would also be in agreement with the results from animal studies (17,98,172,177,210).

iv) Cardiac Function. Ventricular function may be enhanced by increasing the intrinsic contractile properties of the myocardium or the response to inotropic stimulation, as well as by adaptations of extramyocardial factors that have a secondary effect on ventricular performance (24). Exercise training has been shown to alter cardiac dimensions, as well as autonomic state, preload, and afterload, making it difficult to separate cardiac from extracardiac effects of training on ventricular performance.

Numerous animal studies have used a variety of experimental preparations, (including isolated papillary muscles and perfused hearts, and intact anesthetized and awake chronically instrumented animals) to analze the effect of training on intrinsic myocardial contractile properties. Nutter and Fuller (173) reviewed six studies on the effect of training on the mechanical performance of isolated left ventricular papillary muscles. They found that with training, diastolic myocardial length tension relationships did not change. Some studies found evidence of improved contractile performance, while others failed to demonstrate an effect of training on intrinsic contractile function. Isometric or isotonic contractile performance was increased in two series, decreased in two, and showed no change in two. These findings are consistant with other investigations, which have reported variable results (209,213).

Scheuer and associates (19,209,210,211) have systematically studied the adaptations to different forms of training (i.e. running vs swimming in male and female rats), and concluded that both activities produce skelatal muscle adaptations (i.e. increased cytochrome oxidative activity) in male and female rats. However, absolute increases in heart-weight relative to weight-matched control rats occur only in the female swimmers. They found evidence for improved contractile performance (measurements normalized with respect to heart-weight and obtained at several levels of left ventricular filling pressure) in male and female swimmers and in male runners. These findings correlated closely with increased calcium binding in isolated sacroplasmic reticulum and increased actomysin ATPase (210). However, Fuller and Nutter (74), who studied the effect of running in male rats essentially replicated the methods of Scheuer and collaborators, but were unable to demonstrate any training effect on contractility.

There is comparatively little information regarding changes in left ventricle performace in healthy individuals following aerobic training, other than the previously documented increase in maximal CO. Echocardiographic studies have shown no evidence of a significant training effect on intrinsic myocardial contractile performance (24,182). However, most of the reviewed studies were performed under resting conditions. When studying cardiac function it is important to apply stress to the heart since potential differences in intrinsic mechanical performance may not be obvious at rest (213).

Stein et al (225) evaluated the effects of upright interval cycle training on left ventricular function during submaximal supine cycle exercise. After training, exercise was associated with an increase in SV attributable to a decrease in end-systolic dimensions and no change in LVEDD, measured by echocardiography. The investigators interpreted their findings as being indicative of a training-induced enhancement of contractile state during exercise. Rerych et al (190) studied the effects of 6 months of swim training on left ventricular function during upright cycle exercise using first pass radionuclide angiography. After training, CO at peak exercise increased from 25.5 to 32.0 $l \cdot min^{-1}$. This increase in maximal CO was associated with small decreases in exercise HR and a large increase in LVEDV (166 to 204 ml). These data (190) suggest that the dimensional changes often reported by echocardiography may allow for the support of an increased SV via the Frank-Starling mechanism after training. The enhanced SV during exercise was also associated with a greater change in LVESV from rest to peak exercise after training and a lower MAP at peak exercise, suggesting the possibility that the decreased afterload also contributed to the increased output.

Both studies may be criticized on methodological grounds in that the posture and/or mode of training was not well matched to the posture and mode of testing (225). Likewise the selection of competitive swimmers by Rerych et al (190) hardly enhances the applicability of this study to the usual context of training. Thus, dispite the large number of studies in both animals and human, no consistant significant training effect on contractile function has been demonstrated, and the overall effect of exercise training on intrinsic myocardial contractility remains uncertain.

v) Extracardiac Effects. There are only a few studies on the effect of training on venous return and preload, but the data suggest an increase in preload during exercise after training (213). Changes in VO_2 max are parelleled by small but significant changes in total blood volume, mostly accounted for by an increase in plasma volume (24,67,206,213). Studies during maximal exercise following volume loading provide interesting data on the physiology of ventricular filling. Blomqvist and Saltin (24) studied the combined data from several studies (67,194) investigating the cardiovascular response to acute blood volume increases and noted a relationship between maximal SV prior to volume loading and the magnitude of the increase in SV after blood volume loading. They speculated that endurance training alters the ventricular compliance characteristics by modifying right/left ventricular pericardium interaction, resulting in an increase in diastolic reserve capacity. Consistent with this suggestion is the observation of an increased effective left ventricular diastolic compliance due to pericardial rather than myocardial adaptations in studies of chronic volume overload (134).

Afterload reduction secondary to a marked increase in vascular conductance of active skeletal muscle is a critical component of the cardiovascular response to exercise training (24). A marked decrease in systemic vascular resistance facilitates the large increase in SV and CO attained during exercise in trained individuals. Without a marked decrease in afterload the increase in CO could not be achieved, since MAP would be prohibitively increased. In this regard, a strong correlation between the increase in VO_2 max and the decrease in systemic vascular resistance as a result of exercise training has been demonstrated (35)

Local adaptations with increased vascular conductance are prerequisites for effective utilization of a training induced increase in cardiac capacity, due to the inverse relationship between SV and afterload (35). The primary mechanisms responsible for the reduction in systemic vascular resistance with training are poorly defined. They are likely to affect the arterioles and to be regulatory rather than anatomical (227). Training studies in which individuals trained only one leg (48,204) or both legs seperately (127), support the concept that training causes an increase in maximal vascular conductance of working skeletal muscle. The fact that the increases were limited to the trained limb, or limbs, suggests that the change in vascular conductance is mediated primarily by local rather than systemic mechanisms.

The extent to which the increase in the capacity to vasodilate can be translated into an increase in VO_2 max is modified by an opposing vasoconstrictor drive. The strength of this drive is determined by the relationship between systemic oxygen demand, and transport capacity. Clausen (35) has shown that during heavy exercise with large muscle groups, the sympathetic vasoconstrictor activity is capable of overriding the metabolic vasodilation and reduce muscle blood flow. This allows full utilization of the skeletal muscle potential for increased maximum vasodilation, while assuring that the vasodilation does not exceed the pumping capacity of the heart, thus preventing hypotension. Changes in systemic oxygen transport capacity and the systemic impact of the local vascular adaptations are therefore interdependent.

Viewed in the context of potentially greater overall left ventricular dimensions (54,63,97,99) and a greater blood volume after training (20,109) it could be suggested that the greater muscular power and blood volume allow for a greater venous return to the heart. The larger and potentially more complient ventricle allows the heart to operate at a higher volume and to benefit from the Frank-Staring mechanism. The greater demand on the myocardium created by this situation may be tolerated by virtue of the increased myocardial mass following training, as well as the potential reductions in afterload. The increased myocardial mass allows for accommodation to the increase in work and is reflected by the only possible adaptation, an increase in LVEF and a decrease in LVEDSV.

vi) Autonomic Adaptations. Exercise training induces adaptive changes that are apparent at rest and during exercise. In Scheuer and Tipton (213) ample evidence is presented that after exercise training there is an increased parasympathetic activity causing bradycardia. Animal studies noting an increase in myocardial acetylcholine and atrial choline acetyltransferease activity after training suggest an increased availability of acetylcholine at the receptor level (213). During exercise, enhanced parasympathetic tone may still be important at low workloads, and heart rates. However, complete vagal blockade in humans will produce a heart rate of about 130 $b \cdot min^{-1}$ (24). Any increase above this level must be mediated by beta-adrenergic mechanisms.

The response of the sympathetic nervous system to exercise is also affected by training (33). The effect of increased sympathetic activity depends on the concentration of catacholamines as well as the tissue responsiveness. At rest, following training, there is no change in myocardial tissue concentrations or in plasma levels of epinephrine or norepenephrine (34). During exercise, plasma concentrations of both epinephrine and norepinephrine increase with the intensity and duration of the exercise (34). Following training plasma concentrations are lower at any absolute submaximal workload, however no differences were reported at the same relative workload (24). The sensitivity of the sinoatrial node to sympathetic stimulation is unaltered after training (245). In addition training does not appear to change the number of beta-adrenergic receptors in the heart (166). Thus, there is evidence for a decreased sympathetic response to exercise after training.

vii) Skeletal Muscle Adaptations. The most important biochemical adaptation of skeletal muscle to aerobic training is an augmentation of respiratory capacity. This adaptation has been studied most extensively in animal models and confirmed by human muscle biopsy studies. Aerobic training results in increases in the mitiochondrial respiratory chain enzymes involved in oxidation of NADH and succinate (18,105), the enzymes of the citrate cycle (83,107), mitochondrial ATPase, which catalzes the oxidative phosphorylation of ADP to ATP (176); the mitochondrial enzymes involved in the activation, transport, and beta-oxidation of fatty acids (213); and the enzymes of the malate-aspartate shuttle system (105). The increases in the levels of activities of a wide range of mitochondrial enzymes is due to an increase in enzyme protiens (105). This is evidenced by an increase in the protein content of the mitochondrial fraction obtained from skeletal muscle and in the concentration of the cytochromes (107,108).

Electron-microscopic studies on human and rat skeletal muscle have provided evidence that both the size and number of muscle mitochondria increase in response to aerobic exercise training (84,105). Further, aerobic training induces alterations in mitochondrial composition which tend to make skeletal muscle mitochondria more like heart mitochondria in their enzyme pattern (106). In contrast to skeletal muscle, heart muscle, which is continually active, does not undergo an increase in respiratory capacity in response to aerobic training (177,178). The increase in the mitochondrial marker cytochrome c is preceded by an increase in beta-aminolevulinic acid synthetase activity (29). Beta-aminolevulinic acid synthetase is the rate limiting enzyme in Heme synthesis, and has been proposed as an increase in this enzyme plays a key role in the increase in muscle mitochondria in reponse to aerobic training (29,230).

Another biochemical adaptation to aerobic training which may have important physiological consequences is an increase in muscle myoglobin concentration (105). Myoglobin increases the rate of oxygen diffusion though a fluid layer, and may facilitate oxygen utilization in muscle by increasing the rate of O_2 transport though the cytoplasm to the mitochondria.

One consequence of the adaptations induced by aerobic training is that the same work rate requires a smaller percentage of the muscles' maximum respiratory capacity, consequently there is less disruption of homeostasis (105). A second consequence is increased utilization of fat, with a proportional decrease in carbohydrate utilization, during submaximal exercise. The metabolic consequences of these adaptations could play important roles in the increase in endurance and the ability to exercise at a higher percentage of VO_2 max in the trained state, by slowing glycogen depletion and reducing lactate production.

While the increased oxidative potential in muscle after cycle training is well established the data has been less consistant for from strength trained muscle (201). Both Lewis et al (135), and Houston et al (110) noted no change in the potential for supplying energy by aerobic pathways following strength training. Other studies (44,87,130) have reported an increase in SDH activity following strength training. The discrepancy is likely to have been a result of the specific type of the contraction employed in the training program (intensity and duration). There is however, general agreement that the activities of PFK and LDH are not affected, suggesting that strength training does not affect the muscle's potential for resynthesizing ATP via aerobic or anaerobic glycogenolysis or glycolysis.

viii) Summary. In healthy individuals aerobic training changes the composition of cardiovascular response to submaximal exercise, and increases VO_2 max. After training, at a given absolute submaximal workload, the metabolically controlled vasodilation in active skeletal muscles, as well as the neurogenically controlled increase in HR, and peripheral vasoconstriction are less pronounced. Thus, there is a reduction in total muscle blood flow and a relative increase in flow to the non-exercising tissues, with an unchanged CO. The decrease in blood flow to the exercising muscles, HR, and in peripheral vasoconstriction is confined to the trained muscles and is probobly mediated by capillarization and oxidative enzymatic adaptations in the skeletal muscle (8,82).

3.2 Cardiac Patients

i) Oxygen Uptake. Given that improved exercise tolerance is the practical foundation of exercise rehabilitation programs, it is understandable that changes in functional capacity are among the most frequently reported outcomes of these programs. Early studies focused on the the improved exercise tolerance of patients recovering from myocardial infarction (MI), or with stable angina (35,56). More recent studies have focused on either earlier or more aggressive use of exercise (52,61,62).

Beginning in the late 1960s, another group of patients with coronary artery disease began to present themselves for rehabilitation. They were patients recovering from coronary artery bypass surgery (CABS). Over the ensuing two decades there has been a general perception that these patients are more stable, and that their exercise training may progress more rapidly, than post-MI patients or those with chronic stable angina (152,188). This belief relates to the reversal of exercise-induced myocardial ischemia and left ventricular dysfunction following surgery (150,152,188). However, comparison studies on the changes in functional capacity in surgical and medical patients consequent to exercise training is difficult because there is considerable variability (and often mixing of patient types) in several pertinent factors, including type of ergometry, criteria for functional capacity (usually estimated VO_2 max or METs), length of the training program, and particularly, time from the acute clinical eposode to the start of training (117,174).

One study (92) which did evaluate changes in estimated VO_2 max consequent to 3-6 months of aerobic training in groups of post-MI and post-CABS patients noted that the magnitude of the increase in functional capacity was not significantly different for the two groups. The improvement in estimated VO_2 max was similar to that observed in other post-CABS patients (68,174) and medically treated patients (41,235), but considerably less than the 40% increase in measured VO_2 max that was achieved with prolonged, severe exercise training (61,89).

A randomized controlled study, into the efficacy of aerobic training in cardiac rehabilitation, was conducted by Froelicher et al (73) who compared the responses to exercise in a mixed group of patients (MI, angina, CABS). Those assigned to training participated in a conventional exercise training program (i.e. 3 times weekly, aerobic activity) for 1 year. Their results were compared to those from a non-exercising control group. The exercise group demonstrated an increase in both measured (9%) and estimated (12%) VO_2 max, over the same period the control group failed to demonstrate any change in VO_2 max.

Foster et al (68) evaluated changes in the functional capacity of post-CABS patients during a 6 month exercise program which began within 2 weeks following surgery. Both the control group, and the training group demonstrated substantial increases in functional capacity (50% and 85% respectively). The large percentage increases in functional capacity were primarily related to the very low values at the initial testing. The increases in functional capacity during the interval from 2 to 6 months post-surgery was 10 and 20% for the experimental and control groups respectively, which is comparable to those observed in other studies (92,174). These findings suggest that training studies should commence a minimum of eight weeks after surgery, in order to ensure adequate time for recovery from surgery. This suggestion is consistant with the observation that most CABS patients experience a substantial increase in functional capacity during the initial 8 weeks post-surgery, without participating in an exercise program (174,241).

ii) Cardiac Output. A number of studies have examained the CO response to aerobic training in patients with coronary artery disease. The early studies (57,72,196), suggested that the improvements in VO_2 max, seen in cardiac patients, was due to peripheral adaptations which resulted in a slower HR, a lower blood pressure, a reduction in systemic vascular resistance and a widening of the $a\text{-}vO_2$ diff. For instance, Detry et al (57), noted that the SV was unchanged at two submaximal workloads following training, whereas MAP and stroke work were both reduced. However, these studies (57,72,196) have been critisized on methodological grounds, in that they failed to include studies at maximal exercise (57), used a poorly matched control group (72), or a cross sectional design (196). More contemporary studies by Cobb et al (41) and Paterson et al (179) have reinforced the earlier suggestions of little or no change in the exercise CO and SV in patients with ischemic heart disease. Both of these studies suggested that a widening of the $a\text{-}vO_2$ diff was of primary importance to the increased VO_2 max following training in this population.

The model of $a\text{-}vO_2$ diff as the primary determinant of the training-induced changes in VO_2 max in cardiac patients is challenged, however, by the late findings (6 months to 1 year) of Paterson et al (179), the results from Hildenman and Wallace, (103), and by the high-intensity exercise study by Hagberg et al (89). In these studies a substantial increase in SV at a fixed submaximal workload was observed, suggesting that a direct central circulatory adaptation to training in cardaic patients is possible. Hagberg et al (89), reported an 18% increase in SV, whereas MAP was unchanged, consequently stroke work increased suggesting cardiac adaptation. Hildenman and Wallace (103) observed an increase in resting LVEDV, following a 6 month training program conducted 5 days per week. A given CO was achieved by a greater SV and a lower HR, associated with a reduction in LVESV and an increased LVEF. At maximal exercise the patients achieved a higher workload and CO, which was due to both an increased HR and SV. In these three studies, (89,103,179) the increase in SV was associated with relatively high intensity training programs and was attributed to improved myocardial contractility. Unfortunately the studies by Hildenman and Wallace (103), and Paterson et al (179), did not report MAP making it impossible to determine whether the increase in SV was due to a reduced afterload. Also, the use of submaximal studies to predict maximal cardiovascular responses is a technique of questionable value (95,196). However, these findings suggest that the fundamental response to training in some cardiac patients may be more similar to that of healthy individuals than previously believed.

iii) Cardiac Dimensions. Studies on the effect of training on the left ventricular structure of cardiac patients have yielded variable results. Ehsani et al (61) demonstrated significant increases in LVEDD and LVESD, and in posterior wall thickness following 1 year of aerobic training in eight patients with coronary artery disease. The last 9 months of this program were considerable more strenuous than the usual program employed in this patient population, and were sufficient to elecit a 42% increase in VO_2 max. After training, patients exhibited improved left ventricular function as evidenced by a lack of decline in fractional shortening, or mean velocity of circumferential fiber shortening at similar levels of blood pressure attained during isometric exercise. By contrast, Ditchy et al (58) found that the attainment of a training effect (rise in functional capacity from 8.8 to 10.7 METs) in 14 patients was not accompanied by echocardiographic evidence of cardiac adaptations. There were no changes in LVEDD, posterior wall thickness or cross-sectional area. They concluded that the improved functional capacity after training in patients with coronary artery disease was not due to exercise induced left ventricular hypertrophy. The disparate echocardiographic findings in these two studies cannot be related to differences in the pre-and post training levels of aerobic capacity, the duration of training, or the ages of the subjects. In view of the disparity between these studies and the similarity of the results of Ehsani et al (61) to those observed in healthy individuals (54,63,97,99) it has been suggested that the exercise prescriptions employed in conventional cardiac rehabilitation programs is too mild to elecit left ventricular hypertrophy (89).

iv) Cardiac Function. Changes in left ventricular function that allow for an increase in CO and SV relate to the ability to tolerate an elevated LVEDV, without the implications of left ventricular failure (24). Several studies have focused on changes in LVEF during exercise, before and after training. These studies have uniformly reported increases in exercise capacity. However, the LVEF response to aerobic training appears to be quite variable.

Evidence that training may improve myocardial function comes from a study by Jensen et al (115) which showed that during submaximal exercise, that elicited the same MAP and HR, the LVEF was greater (59 vs 55%) after 6 months of training in a phase III type cardiac rehabilitation program. Hildenman and Wallace (103), noted an increase in maximal SV and LVEDV after six months of aerobic training, from rest though to maximal exercise, the LVEF, which fell in all subjects before training, failed to decrease in most subjects post training. Froelicher et al (73) noted a slight increase in SV and CO, but not in LVEF during exercise among their angina-free patients. Cobb et al (41) evaluated the response to six months of aerobic training. Dispite a marked increase in work capacity, no changes in rest or exercise LVEF were evident.

In the study by Foster et al (68) the peak exercise LVEFs actually decreased during the interval from 2 weeks to 2 months postoperatively, although the values were higher than observed preoperatively. In this study, work capacity increased significantly in the training group, whereas changes in LVEF were unrelated to intervention mode. The decreased peak exercise LVEF in this study was associated with conciderable increases in blood pressure during the same interval. This association led the authors to suggest that the very low afterload observed soon after surgery may have produced spurious enhancement of left ventricular function. Results of this investigation suggests that exercise training has little effect on left ventricular function as defined by LVEF. The failure of exercise training to change LVEF is consistant with results reported on normal healthy individuals (191).

v) Summary. Exercise based rehabilitation programs have a definite benificial effect on clinical status, as evidenced by improved work capacity, (52,62) reduced incidence of anginal attacks (189) and indications of a reduced rate of morbidity/mortality (171). Their effect on left ventricular function however, which is an important prognosticator of survival (42,239) is not clear. Verani et al (235) and Ehsani et al (61) have presented evidence that resting left ventricular performance may be improved as a result of training. Jensen et al (115) contends that LVEF may be improved during submaximal exercise, but not under maximal exercise conditions. Conversely Cobb (41), and Foster (68), have demonstrated that LVEF does not change as a result of training, dispite large increases in physical work capacity. The response to exercise training in cardiac patients would appear to be fundamentally similar to that in healthy individuals, differences may be attributable to the training intensities, and to intrinsic differences attributable to myocardial ischemia, or scar (23).

4. CIRCUIT WEIGHT TRAINING

i) Oxygen Uptake. While there is little doubt that CWT will improve muscular strength and endurance (5,75,247), the cardiovascular responses to such training remain controversial. Allen et al (5), were perhaps the first to study the effect of CWt on aerobic power.

Their 12 week program, which consisted of a series of 30s of high resistance, low repetition work bouts followed by 60s relief intervals, failed to effect VO_2 max. This finding, combined with the earlier work of Nagle and Irwin (170), suggest that traditional weight training with heavy weights is too intermittent to enhance aerobic power.

The effects of CWT, with timed work and relief intervals, was studied by Wilmore et al (246). The subjects in this study exercised at 40-55% of 1RM, executing as many repetitions as possible during 30s work interval, with a 2:1 work to relief ratio. After training thrice weekly for 10 weeks the women demonstrated a 11% improvement in aerobic power however, the men failed to demonstrate any improvement. The authors reasoned that differences in initial fitness, affected the relative training intensities, suggesting that CWT is an effective activity for deconditioned individuals.

Gettman et al (78), conducted a CWT study based on the premise that a larger training effect would be observed after a longer program. However, following a 20 week program, which incorporated 10 training stations, relief interval of 30 to 20s, and an exercise intensity of 50% of 1RM, the improvement in aerobic power was only 3.5%. The changes in aerobic power, observed with CWT were considerably lower than the 15% improvement in VO_2 max observed following a 20 week running program conducted simultaneously on another group of 16 men. In a subsequent study however, Gettman et al (75), reported a 12 and 13% increase in aerobic power, for men and women repectively who completed a 20 week CWT program. The training program was similar to a program used in the previous study (78) with several important modifications. The program incorporated 30s work intervals and a 1:1 work to relief ratio, unlike the earlier study where no restrictions were placed on the exercise intervals. Secondly, three circuits were completed as opposed to only two in the previous study. As a result, the rate of work, and accummulated work per session were probably increased, thus accounting for the enhanced aerobic power.

The impact of accumulated work on the efficacy of CWT is discussed by Gettman and Ayres (79). Following 10 weeks of isokinetic circuit training, a slow velocity group (trained at 1.04 rad·s^{-1}) demonstrated a 10% increase* in VO_2 max, whereas subjects trained at a faster velocity (2.08 rad·s^{-1}) improved by only 3%. The authors suggested that, because the number of repetitions per set were constant for the two groups, the slow-velocity group averaged more time per workout and accummulated more work per training session. This suggests that total exercise time and work accommplished per session help determine the extent of aerobic improvemnet with CWT (76).

One of the problems of evaluating the efficacy of CWT on cardiovascular parameters relates to the specificity of training. Changes from training are specific to the muscles involved and the pattern in which they are used (35). Thus the validity of using a treadmill or cycle ergometer to evaluate the cardiovascular response to CWT may be questioned. However, to date, only two groups have examined this issue. Allen et al (5), assessed VO_2 max during arm work as well as leg work, whereas Gettman et al (78) incorporated two nonspecific tests into an examination of the cardiovascular impact of CWT. In both instances, variations in the test protocols did not alter the relative results (76).

Gettman et al (77), examined the interplay of CWT with aerobic training. The study had two purposes: to compare the physiological effects of CWT and running; and to evaluate CWT after training by running. The initial eight weeks of CWT using isokinetic devices resulted in a 3% increase in VO_2 max. The subsequent eight weeks running program, at 85% of maximal HR, resulted in an additional 8% improvement. At the completion of the running program half the men returned to the CWT program, whereas the rest continued running. Both groups maintained aerobic power equally well for the final eight weeks of the study. Several practial implications were drawn from this study. First, CWT could be used as a means of maintaining aerobic power. Secondly, no muscular soreness was observed during CWT using isokinetic devices. This is thought to be due to the absence of eccentric contractions (231). Consequently, it was suggested that circuit training using devices that allow concentric only exercise might be useful as a starting exercise program.

The controversy regarding the efficacy of CWT on cardiovascular fitness may be related to the relative exercise intensity of the various programs. In order to enhance cardiovascular fitness, the American College of Sport Medicine (6) recommends that the training intensity should be equivalent to 60-90% of maximal HR reserve, or 50-85% of VO_2 max. The unique feature of CWT is the potential differentiation between the cardiovascular and metabolic stimuli for altering VO_2 max. During CWT an individual may work in excess of 80% of maximal HR, but at less than 50% VO_2 max (5,94,112). This, suggests that systemic effects are unlikely to develop if the program is carried out at low levels of VO_2.

McArdle and Foglia (147) conducted one of the earliest studies on the energy cost of weight training exercises. Four weight training exercises of eight repetitions each were studied, the peak metabolic intensity was 25 $kJ \cdot min^{-1}$. The author concluded that the energy expenditure attained during weight training provided only a moderate stress for improving aerobic power. Wilmore et al (247) reported the energy cost of CWT using Universal Gym equipment averaged 37.7 and 25.5 $kJ \cdot min^{-1}$ for the men and women respectively. The men in the study, worked at an average of 74% of maximal HR and 39% VO_2 max, whereas the women averaged 84% of their maximum HR and 45% VO_2 max.

Hempel and Wells (94) noted that the energy cost of strength training with Nautilus equipment averaged 21.5 $kJ \cdot min^{-1}$ and 32.7 $kJ \cdot min^{-1}$ for men and women respectively. The values for percent VO_2 max used during the circuit averaged 35.9 for the women and 38.3 for the men. The corresponding HR responses to the training program averaged 73 and 77.5% for the women and men respectively. The authors concluded that CWT using Nautilus equipment would not stimulate metabolic responses to the extent indicated by the the HR reponse, and that Nautilus circuit training elecits only a marginal aerobic exercise intensity. The results from Hurley et al (113) supports this claim. In this particular study young healthy subjects completed a 16 week CWT using Nautilus equipment. The HR response to the training exceeded 80% of the HR reserve, however, the VO_2 represented approximately 45% of VO_2 max. No significant changes in VO_2 max were observed over the study period.

The Disproportionate increase in HR, relative to VO_2 may be attributed to greater sympathetic stimulation during CWT (112). During CWT, approximately 50% of the exercises involve the upper extremety exercise. As discussed previously, arm work is associated with elevated sympathetic stimulation, and HR at a given VO_2 (12,49).

Gettman et al (80) reported that the energy costs of circuit training using isokinetic devices averaged 41 $kJ \cdot min^{-1}$ with the HR equivalent ot 69% of maximum, and VO_2 at 49% of VO_2 max. Katch et al (118) reported that the energy expenditure of hydraulic resistance exercise averaged 29.4 and 11.5% greater than CWT on Nautilus and Universal Gym equipment respectively. The HR response to the 20s work intervals and 1:1 work to relief ratio averaged 85% of maximum while the corresponding value for VO_2 was 53% of VO_2 max. Combined these studies (80,118) suggest that the HR and metabolic response to concentric-only exercise for the agonist and antagonist muscles involved in a given movement achieves a level necessary to promote improvement in aerobic power.

ii) Cardiac Output. Several studies have examined both resting HR and the HR response to submaximal aerobic exercise following short term weight training programs. Papers by Allen et al (5) and Hurley et al (112) reported that the hemodynamic response to submaximal exercise on a bicycle (5) or a treadmill (112) is unchanged following CWT. In contrast, papers by Stone et al (228) and Laird et al (131) noted that both the resting HR and the HR response to a given submaximal workload were reduced following short term weight training programs.

Kanakis and Hickson (116) reported a 12% decrease in resting HR following 10 weeks of weight training. The SV was increased slightly however no changes in LVEDD or LVESD were noted. Further, Gettman et al 1982, demonstrated an increase in both VO_2 max and maximum O_2 pulse following a 12 week CWT program. Astrand and Rodahl (12) explain that O_2 pulse is an indirect measure of SV. This implies that increase in VO_2 max noted by Gettman et al (75) may have been due to an increased SV. Thus, information on changes in SV consequent to CWT would be valuable in delineating the underlying mechanisms responsible for changes in maximal aerobic power.

iii) Muscular Strength and Endurance. Investigators who used the 1RM test for the assessment of strength have reported improvements in leg press or bench press strength ranging from 7 to 44%, following a program of CWT (75,76,112).

The study by Gettman et al (78), utilized an isokinetic device to assess changes in muscular strength consequent to a 20 week training program. Those subjects allocated to CWT demonstrated a 43% increase in strength following training. The result observed in the CWT group was significanty greater than those observed in either a control group or aerobic trained group. The program of isokinetic circuit training conducted by Gettman et al (77), resulted in an average 31% increase in muscular strength, assessed via isokinetic bench press and leg press exercises. Weltman et al (242) reported an 18-36% increase in isokinetic concentric work among pre-pubertal boys who completed a 14 week CWT program using hydraulic resistance equipment. Thus, a CWT program can be expected to elict increases in muscular strength and endurance.

iv) Cardiac Patients. Only one group has examined the efficacy of a CWT in coronary patients. Keleman et al (122), compared the effects of of a combined jogging/CWT program with those associated with a jogging/volleyball program. The CWT incorporated 10 exercise stations (Universal Gym variable resistance equipment). Each exercise was performed at 40% of 1RM with 30 second work intervals and a 1:1 work to releif ratio. Upon completion of training the CWT group demonstrated a 12% increase in exercise treadmill time, and a mean 24% increase in 1RM strength. The jogging/volleyball group failed to demonstrate any change in either treadmill time or 1RM strength. When the patients were stratified on the basis of myocardial infarction, CABS, and left ventricular dysfunction, no significant differences in the magnitude of the aerobic or strength improvement was observed. While these results are encouraging, in that they suggest that CWT may be a viable adjunct to traditional walk/jog programs, the compliance of the CWT and volleyball groups, to their respective programs may have biased the results in favour of the CWT. Further, this study used maximum treadmill time an the index of cardiovascular fitness, however performance time on a treadmill may not necessarily relate highly to VO_2 max. (73).

SUMMARY

As discussed, variable results have been reported concerning the details of the central and peripheral circulatory adaptations to training in patients with coronary artery disease. Despite the lack of clear cut evidence of a beneficial cardiac effect, (7,212) aerobic exercise remains the exercise of choice for most rehabilitation programs. This type of exercise places a heavy demand on the central circulation. Patients with severe coronary artery disease may be unable to tolerate vigorous aerobic exercise because of symptoms. They are also at risk of ischemic induced arrhythmias while engaging in endurance type activities (35). Thus it would be of great benefit if the training effects could be achieved without the high central circulatory demands associated with aerobic exercise.

It is suggested that circuit training using hydraulic resistance equipment might be useful for patients with low exercise tolerance, or ventricular dysfunction. By training relatively small muscle groups one at a time, the training adaptations may be achieved without precipitating a sustained heavy load on the central circulation. In addition to the clinical interests, it is also of physiological interest to evaluate the extent to which the cardiovascular adaptations can be achieved by training which does not produce a sustained heavy demand on the central circulation.

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APPENDIX B

DEFENITION OF TERMS

Afterload: the resistance against which the heart contracts. Afterload describes the external forces that oppose shortening and ventricular ejection, including ventricular end-diastolic volume, aortic impedance and sytemic vascular resistance.

Circuit weight training (CWT): a form of strength training in which different exercises are performed with resistance equipment in a pre-established exercise to rest sequence. The typical CWT program consits of 8 to 12 exercise stations. Ten to 20 repetitions are usually perfromed at each station, followed by a relief interval (no exercise) of between 20s and 1 minute.

Constant resistance: maintained resistance at a stable level, although the muscle's force generating capacity can vary conciderably thoughout the range of motion.

Diastolic reserve: represents the increment in fiber shortening due to an increase in fiber length (i.e. increased ventricular filling). The limit to the diastolic reserve is set by pulmonary venous pressure.

Echocardiography: the transmission of pulsed ultrasound though the heart and the detection of the returning echos to detail the position and movement of the cardiac acoustic interfaces. This technique can define intracardiac structures and their motion and can be used to measure ventricular cavity size, shape and wall thickness.

Ejection Fraction (LVEF): the percentage of left ventricular end diastolic volume (LVEDV) pumped from the ventricle.

Frank-Starling mechanism: the increased contraction of the heart muscle caused by stretching of the muscle fibers upon increased filling of the cardiac chambers.

Hydraulic Circuit Training (HCT): A circuit training program using variable hydraulic cylinders to provide the resistance to movement. These hydraulic cylinders allow for double concentric, bidirectional muscle work.

Isokinetic contractions: refers to muscular contractions performed against a resistance that moves at a preset constant velocity. Such contractions enable muscle to mobilize their maximum tension-generating capacity thoughout the full range of motion.

Isometric contractions: static muscular contractions with no visible movement. The limb is isolated at a particular joint angle. Tension is developed but no mechanical work is performed.

Isotonic contractions: dynamic muscular contractions that are either eccentric (lengthening) or concentric (shortening). Mechanical work is performed as the muscle shortens or lengthens.

LaPlace law: tension in the vessel wall equals the transmural pressure multiplied by the radius of the vessel divided by the wall thickness

Left Ventricyular End-Diastolic Volume (LVEDV): volume of blood contained in the left ventricle at end diastole.

Left Ventricyular End-Systolic Volume (LVESV): volume of blood contained in the left ventricle at end systole.

Maximal voluntary contraction (MVC): maximum force that can be generated in a single contraction.

Preload; the end diastolic fiber length, or the extent of ventricular filling before contraction. May be altered by changes in blood volume, venous tone, myocardial contractility, and/or ventricular compliance.

Pulmonary Wedge Pressure: pressure measured by a catheter wedged in a small branch artery of the pulmonary artery tree. A reflection of left atrial and left ventricular end diastolic pressure.

Radionuclide ventriculography: the assessment of cardiac performance, at rest or with interventions, by intravenous radioisotope injection. There is analysis of first transit though the central circulation (first pass technique) or of the entire blood pool after intravascular labeling (equilibrium gated technique).

Rate pressure product (RPP): an index of myocardial oxygen consumption. The product of heart rate and systolic blood pressure.

Systolic reserve: represents the increase in fiber shortening due to increaments in the contractile state and a reduction in afterload

Wall tension: intramyocardial wall tension is proportional to the product of ventricular volume (radius). Increased systolic wall tension increases myocardial oxygen demand.

Weight training: refers to a system of strength training in which the muscles exert tension or force to overcome a fixed or variable resistance during the contraction.

APPENDIX C
PHYSICAL EXAMINATION FORM

NAME:____________________________ AGE:_____ SEX:_____ D.O.B.:________

ADDRESS:____

PHONE: (H) __________ (W)__________ OCCUPATION: ___________________

HABITUAL PHYSICAL ACTIVITY: sedentary (no regular exercise) _____
moderate (1-2 days/week @ THR) _____
regular (>3days/week @ THR) _____

SMOKING: Yes ___ No ___ Quit ___
Smoked for _____ years. Quit _____ years ago.
Number/day _____

ALCOHOL: drinks/week; beer ___ wine ___ liquor ___

PREVIOUS MEDICAL HISTORY:____

____

FAMILY HISTORY OF:
Coronary artery disease ____

Hypertension ____

Stroke ____

Diabetes ____

SYSTEMIC ENQUIRY: C.N.S.
C.V.S.
R.S.
G.I.
G.U.
Gyn.
Psych.

EXAMINATION: Pulse ________________ Regular ________________

Blood pressure ________
C.V.S. ( )
R.S. ( ) CHECK IF OK
Abd. ( )
C.N.S. ( )

Comments:____

____

APPENDIX D

CURRICULUM VITAE

NAME: Robert Gerald Haennel

ADDRESS:
10726 67 Ave.
Edmonton Alberta
T6H 1Z9

DATE OF BIRTH: August 31, 1954

CITIZENSHIP: Canadian

MARITAL STATUS: Married; 2 children

ACADEMIC QUALIFICATIONS:

INSTITUTION	YEAR OF COMPLETION	DEGREE
University of Alberta	1987	Ph.D. Physical Education and Sport Studies. Specialized in Exercise Physiology and Cardiac Rehabilitation.
University of Western Ontario	1980	Masters Degree in Physical Education. Specialized in Exercise Physiology.
University of Western Ontario	1977	Honours Bachelor degree in Physical Education.

PROFESSIONAL EXPERIENCES:

Post-Doctoral Fellow, Division of Cardiology, University of Alberta. Investigating the physiological adaptations of patients with congestive heart failure to various exercise modalities. Co-ordinating a placebo-controlled efficacy study of Carwin (Xamoterol) in heart failure patients. Also working as a sessional lecturer in the Department of Physical Education and Sport Studies Education. June 1987-present.

Research Fellow, -Division of Cardiology, University of Alberta. Responsible for several projects investigating the efficacy of various exercise training programs for post-myocardial infarction patients. May 1986 to June 1987.

Coordinator, University of Alberta Fitness Unit. This positions' prime responsibility involved the development and implementation of the Fitness Appraiser Certification and Accreditaion program in Alberta. Further duties included assisting in the day to day operations of the fitness testing and lifestyle center, and the development of materials for the provincial resource center.
May 1982 to April 1986.

Exercise Specialist, Alberta Hospital, Edmonton, Alberta. Developed and implemented a Health-Fitness program, for various services within the hospital. Direct programming responsibilities included the implementation of a physical fitness appraisal program, and the establishment of related exercise programs for psychiatric patients.
September 1981 to December 1982.

Research Associate, Community Recration Development Division, Alberta Recreation and Parks, Edmonton, Alberta. Researched and developed a technical planning manual. Further responsibilities included assessing the consultants' evaluation of provincially funded Health-Fitness programs and developing and executive summaries for these reports. January 1981 to July 1981.

Exercise Specialist, Edmonton Cardiac Instiute, Royal Alexandra Hospital, Edmonton, Alberta. Conducted graded exercise tests. Established and monitored exercise prescriptions, and supervised technical staff. Further responsibilities included preparation of educational materials, and seminars for the patients.
September 1979 to December 1980.

Program Director, YM-YWCA, Moose-Jaw, Saskatchewan. Responsible for the administration of all Fitness and Health programs within the organization. Implimented special programs for post-cardiac patients, and employee groups. Additional responsibilities included the development and supervision of a fitness instructor training program, and a fitness appraisal unit. July 1978 to August 1979.

Teaching Assistant, Faculty of Physical Education University of Western Ontario. Instructed fitness classes in Cardiac Rehabilitation and conducted seminars in Scientific Foundations of Health. September 1977 to May 1978.

Physical Fitness Consultant, London Fitness Foundation, London, Ontario. Conducted a fitness appraisals and exercise programs for a variety of employee groups. May 1977 to September 1977.

PUBLICATIONS:

ARTICLES:

Haennel, R.G., K.K. Teo, H.A. Quinney, and C.T. Kappagoda. The effects of hydraulic circuit training on cardiovascular function. Medicine and Science in Sport and Exercise. Submitted, 1988.

Haennel R.G., K.K. Teo, P.V. Greenwood, G.D. Snydmiller, M.D. Hetherington, H.A. Quinney, and C.T. Kappagoda. Changes in cardiac output and blood pressure during to isokinetic exercise. European Journal of Applied Physiology, Submitted, 1987.

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Hetherington D.M., K.K. Teo, R.G. Haennel, C.T. Kappaggoda, and R.E. Rossall. Response to upright exercise after myocardial infarction. Cardiovascular Research. 21:399-406, 1987.

Hetherington D.M., R.G. Haennel, K.K. Teo, and C.T. Kappagoda. Importance of considering ventricular function when prescribing exercise after acute myocardial infarction. American Journal of Cardiology. 58: 891-895, 1986.

Hetherington M., K. Teo, R. Haennel, P. Greenwood, R. Rossall, and T. Kappagoda. Use of impedance cardiography in evaluating the exercise response of patients with poor left ventricular function. European Heart Journal. 6: 1016-1024, 1985.

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ABSTRACTS:

Haennel R.G, S.R. Petersen, C.T. Kappagoda, A.N. Belcastro, D.C. Reid, and H.A. Quinney. The influence of high velocity circuit resistance training on aerobic power.
Canadian Association of Sport Sciences Annual Meeting, Ottawa, October, 1986.
Canadian Journal of Appled Sport Sciences. 11: 18P, 1986.

Haennel R.G. K.K. Teo, D.M. Hetherington, P.V. Greenwood, G.D. Snydmiller, H.A. Quinney, and C.T. Kappagoda. Cardiac output and blood pressure changes during high velocity isokinetic exercise.
Graduate Research Symposium, Edmonton, May, 1986.
Conference Proceedings. p.17, 1986.

Teo K.K., A. Suthijumroom, M. Senaratne, R. Haennel, M. Hetherington, E. Ryan, and T. Kappagoda. Abnormal cardiac function in diabetics.
The X World Congress of Cardiology, Washington, September, 1986.

Conference Proceedings, p.289, 1986.

Hetherington M., K.K. Teo, A. Suthijumroom, M. Seneratne, R. Haennel, E. Ryan, and T. Kappagoda. Abnormalities of the exercise stroke volume-heart rate relationship in diabetics.
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Clinical Investigative Medicine. 9(3): A41, 1986.

Haennel R.G., K.K. Teo, D.M. Hetherington, P.V. Greenwood, G.D. Snydmiller, H.A. Quinney, and C.T. Kappagoda. The cardiovascular response to isokinetic exericse.
American Physiological Society Fall Meeting, Niagra Falls, October, 1985.
Physiologist. 28: 300, 1985.

Haennel R.G., K.K. Teo, M.D. Hetherington, P.V. Greenwood, G.D. Snydmiller, C.T. Kappagoda and H.A. Quinney. The effects of a 10 week hydraulic resistance training program on the cardiovascular system.
Canadian Association of Sport Sciences Annual Meeting, Quebec City, October, 1985.
Canadian Journal of Appled Sport Sciences, 10: 13P, 1985.

Haennel R.G., K.K. Teo, D.M. Hetherington, P.V. Greenwood, G.D. Snydmiller, H.A. Quinney, and C.T. Kappagoda. Acute changes in heart rate stroke volume and cardiac output during isokinetic exericse.
Third World Congress on Cardiac Rehabilitation, Caracus, October 1985.
Conference Proceedings, p.172, 1985.

Teo K.K., R.G. Haennel, D.M. Hetherington, G.D. Snydmiller, P.V. Greenwood, C.T. Kappagoda and H.A. Quinney. Cardiac output and blood pressure changes during high velocity resistance exercise.
Canadian Association of Sport Sciences Annual Meeting, Quebec City, October, 1985.
Canadian Journal of Appled Sport Sciences, 10: 33P, 1985.

Hetherington D.M., R.G. Haennel, K.K. Teo, R.E. Rossall, P.V. Greenwood, C.T. Kappagoda, and H.A. Quinney. Differences in stroke volume during dynamic exercise in normal subjects and post infarction patients.
The III World Congress of Cardiac Rehabilitation, Caracus, October, 1985.
Conference Proceedings, p. 89, 1985.

Hetherington D.M., R.G. Haennel, M. K.K. Teo, R.E. Rossall, P.V. Greenwood, C.T. Kappagoda and H.A. Quinney. Heart rate (HR) and stroke volume (SV) changes during dynamic exercise in normal subjects and post infarction (MI) patients.
Canadian Association of Sport Sciences Annual Meeting, Quebec City, October, 1985.
Canadian Journal of Appled Sport Sciences, 10: 14P, 1985.

Hetherington D.M. K.K. Teo, R.G. Haennel, P.V. Greenwood, R.E. Rossall, and C.T. Kappagoda. Impedance cardiography (IC) as a method for evaluating exercise response in patients with left ventricular dysfunction.
International Symposium on Left Ventricular Dysfunction, Jerusalem, 1985.
Conference Proceedings, p.54, 1985.

Hetherington D.M. K.K. Teo, R.G. Haennel, R.E. Rossall, and C.T. Kappagoda. Identification of subsets of post infarction patients by stroke volume responses to graded exercise.
Inernational Symposium on Left Ventricular Dysfunction, Jerusalem, 1985.
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Hetherington D.M. K.K. Teo, R.G. Haennel, R.E. Rossall, and C.T. Kappagoda. Effects of B-blockade on exercise stroke volume in patients 8 to 10 weeks post infarction
International Symposium on Left Ventricular Dysfunction, Jerusalem, 1985.
Conference Proceedings, p.40, 1985.

Teo K.K., D.M. Hetherington, R.G. Haennel, P.V. Greenwood, and C.T. Kappagoda. Measurement of cardiac output by impedance cardiography during maximal exercise testing.
The III World Congress on Cardiac Rehabilitation, Caracus, October, 1985.
Conference Proceedings, p.136, 1985.

Hetherington M., K. Teo, R. Haennel, R. Rossall, and T. Kappagoda. Post infarction characterization of patient subsets by exercise stroke volume.
Canadian Society for Clinical Investigation Annual Meeting, Vancouver, September 1985.
Clinical and Investigative Medicine, 8: A47,1985
Fifth Annual Heritage Medical Research Days, Calgary, November, 1985.

Hetherington M., K. Teo, R. Haennel, R. Rossall, and T. Kappagoda. Stroke volume (SV), cardiac output (CO) and vascular resistance (SVR) during exercise 8-10 weeks post infarction.
XII Interamerican Congress on Cardiology, Vancouver, June, 1985.
Conference Proceedings, p.136, 1985

Haennel R.G., K.K. Teo, G.D. Snydmiller, H.A. Quinney, and C.T. Kappagoda. The cardiovascular effects of inversion.
Canadian Association of Sport Sciences Annual Meeting, Moncton, Oct.,1984.
Canadian Journal of Applied Sport Sciences, 9: 26P, 1984.

Haennel R. Exercise therapy in hypertension.
Symposium on the Efficacy and Compliance in Hypertension and Renal Failure, Edmonton, November, 1984.
Symposium Proceedings, p.37, 1984.

Teo K., M. Hetherington, R. Haennel, T. Kappagoda, and P. Greenwood. Accuracy of impedance cardiography during maximal exercise testing.
American Physiological Society Fall Meeting, Anaheim, October, 1984.
Physiologist, 23: 250, 1984.
Fourth Annual Heritage Medical Research Days, Edmonton, November, 1984.

Hetherington M., K. Teo, R. Haennel, R. Rossall, and T. Kappagoda. Use of impedance cardiography for evaluating exercise response in patients with poor left ventricular function.
Canadian Society for Clinical Investigation Annual Meeting, Montreal, September, 1984.
Clinical and Investigative Medicine, 7: A32, 1985
Fourth Annual Heritage Medical Research Days, Edmonton, November, 1984.

Snydmiller G., M. Kavanaugh, D. Marshall, R. Haennel, H. Quinney, and T. Kappagoda. Prevalence of ECG abnormalities in a fitness appraisal center.
Canadian Association of Sport Sciences Annual Meeting, Moncton, October, 1984.
Canadian Journal of Applied Sport Science, 9: 41P, 1984.

INVITED PRESENTATIONS:

Role of nueral receptors in the heart rate and blood pressure response to exercise. Certified Fitness Appraiser Seminar, Red Deer, Alberta, January, 1987.

Cardiovascular and Pulmonary Physiology. Certified Fitness Appraiser Course, Blue Lake, Alberta, May, 1986.

Physical Fitness Appraisal for Employees. A Brief Presented to the Solicitors General Department, Government of Alberta, Edmonton, Alberta, April, 1986.

Cardiovascular Regulation During Exercise. Certified Fitness Appraiser Seminar, Red Deer, Alberta, January, 1986.

Exercise Testing. Fitness Round-Up, Banff, Alberta, April, 1985.

Exercise Prescription For Post Myocardial Infarction Patients. Certified Fitness Appraiser Course, Blue Lake, Alberta, May, 1985.

Exercise Training For Hypertension. Nephrology Rounds, University Hospital, Edmonton, Alberta, October, 1984.

Use of ECG's During Exercise Testing. Certified Fitness Appraiser Course, Blue Lake, Alberta, May, 1984.

Exercise Training for Psychiatric Patients. Medical Grand Rounds, University Hospital, London, Ontario, 1980.

SCIENTIFIC ORGANIZATIONS:

Member, Canadian Physiological Society
Member, Canadian Association of Sports Sciences
Member, American Physiological Society
Member, American College of Sports Medicine

PROFESSIONAL CERTIFICATION:

Exercise Program Director (Candidate) American College of Sports Medicine.
Exercise Specialist, American College of Sports Medicine, 1980.
Certified Fitness Appraiser, Canadian Association of Sport Sciences, 1983.
R.F.A. Course Conductor, Canadian Association of Sport Sciences, 1983.
Trainer of Fitness Leaders, Alberta Fitness Leadership Certification Association, 1985.
National Executive Committee, Fitness Appraisal Certification and Accreditation Program, 1985-1986.